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THE JOURNAL OF PERIODONTOLOGY is published to serve as the professional journal of periodontia; to keep dentists in touch with research being carried on in this field and related sciences; to stimulate greater interest in the supporting tissues of the teeth and their relation to oral and general health. The plan to continue the publication of abstracts indefinitely will make the Journal a fund of extensive information, readily available to students, teachers, researchers and clinicians.

The Journal is the official organ of the American Academy of Periodontology and will contain, therefore, those papers of scientific and practical interest which are presented at the annual meetings of that society. The proceedings of the 1936 annual meeting will be published in the January, 1937 issue. This number contains the abstracts of articles on periodontia which appeared during the year of 1935.

We are grateful to all who have made available the subject matter from which the abstracts and copies have been made, including the authors of the original articles, the journals mentioned in the text of the abstract section, and those who have aided in any way in compiling the material presented in this issue.

The importance of a subject in a dental school curriculum should be determined largely on the value to society that ensues from educating students in that subject. Viewed from this angle periodontia is of equal rank with operative dentistry. Adequate preparation of students in periodontia and adequate practice by graduates of this learning will save as many or more teeth than the efficient practice of operative dentistry. All the arguments that apply to adequate preparation and practice of one apply equally well to the other. It is poor consolation to a patient who has to lose his teeth from periodontoclasia that the ravages of decay in his mouth were checked by exquisitely executed operative procedures.

—Excerpt from Article on next page.

THE JOURNAL OF PERIODONTOLOGY

JULY, NINETEEN HUNDRED THIRTY-SIX

The Undergraduate Course in Periodontia

By HAROLD J. LEONARD, D. D. S., B. A.
New York City

THE number of teeth lost due to periodontal disease is actually greater, according to Brekhus, than those lost on account of dental caries. The prevention of gingivitis, periodontoclasia, and the cure of these diseases in their early stages is more positive than the prevention and cure of dental caries. The technics involved for the prevention and treatment of gingivitis and periodontoclasia are not difficult to teach or to learn by the average dental student. There is every reason why these technics and the science on which they are based should be a part of the education and training of all dental students for practice. Yet it is a fact that many of the dental schools either entirely omit periodontia as a subject or place it as a subdivision of Dental Pathology, Dental Therapeutics, or Operative Dentistry where scant attention is given to it. The result is that the majority of students graduating from American Dental Schools, even today, are totally unprepared to deal adequately with periodontoclasia and gingivitis when these almost universal diseases appear in practice.

The importance of a subject in a dental school curriculum should be determined largely on the value to society that ensues from educating students in that subject. Viewed from this angle periodontia is of equal rank with operative dentistry. Adequate preparation of students in periodontia and adequate practice by graduates of this learning will save as many or more teeth than the efficient practice of operative dentistry. All the arguments that apply to adequate preparation and practice of one apply equally well to the other. It is poor consolation to a patient who has to lose his teeth from periodontoclasia that the ravages of decay in his mouth were checked by exquisitely executed operative procedures. It is quite as essential from the standpoint of society that dentists be trained to prevent and cure periodontoclasia as that they be trained to fill teeth well. This means that the same care should be used in the selection of competent teachers and in the instruction of students in periodontia as in the teaching of operative dentistry. It may no more be taught as a minor phase of some other subject by teachers whose interest and training is elsewhere, than opera-

tive dentistry. Periodontia is a subject of major social import in the dental curriculum and must have a recognized place and be taught by teachers specially prepared and devoted to it.

The fact that periodontia is one of the most essential subjects in the preparation of dentists for practice does not mean that it requires as much time as any other subjects of equal rank. The hours required in the dental curriculum for operative dentistry range between five hundred and a thousand in the different schools. Operative Dentistry involves a vast range of theory and practice with large numbers of exact technics. Periodontia is much smaller both in content of theory and in technic. It requires much less time for its presentation and technical training. The criterion for the amount of time required for a subject like periodontia is the least amount necessary to prepare students well for intelligent handling of their cases after graduation; and as a basis for the skill which may only be acquired by extended practice after graduation.

Periodontia is closely tied up with other subjects in dentistry and must be so taught. It is almost impossible to teach it adequately in a school where the teachers of operative dentistry, crown and bridge, and prosthetics are ignorant about periodontia and contemptuous of it, or where the dean of the school sees nothing in this subject. Diseases of the periodontium are of vital importance to all other branches of dentistry. Neglect in teaching periodontia in a dental school is as absurd as neglect in teaching the science of foundation structures and geologic formations in the construction of buildings or bridges in engineering. Without knowledge of the investing tissues of the teeth, the causes of disease in these tissues, and the means of treating disease once it has occurred, the dentist is poorly equipped to practice any type of dentistry.

Far too frequently patients are seen who have been in the hands of conscientious dentists yet must lose teeth because of neglect in consideration of the supporting tissues of the teeth in relation to the operative procedures. Overhanging filling margins that irritate the gingiva lead to periodontal pocket formation and loss of supporting bone. Faulty contacts that permit food impaction are an important source of periodontal disease. Occlusal restorations that are not adjusted to the excursive movements of the jaws bring lateral stresses and movements to the teeth that result in destruction of alveolar bone. Missing teeth permit shifting of adjoining and opposing teeth in a manner that results frequently in wrecking the whole dental mechanism. Clasps on partial dentures designed to move the abutment teeth each time the denture is inserted or removed invariably result in loss of these teeth. Orthodontic appliances so used as to destroy supporting bone and so insanitary that they involve the damaged bone areas in infective inflammation, effectuate ruin of the masticatory mechanism. These plus many more examples which might be cited indicate the interrelation between periodontia and all other fields of dentistry, and show why so much of dental service even when practiced by conscientious dentists results in failure.

Almost anything is possible in the line of esthetic and functional dental rehabilitation providing there are healthy roots well imbedded in healthy bone. If the supporting tissues are diseased and not put in health the best of restorations are but as a house built upon the sand.

The teaching of periodontia must be closely correlated with the teaching of oral pathology, oral hygiene, and oral diagnosis. The art of treating the gingiva and periodontal tissues in order to prevent and cure periodontal diseases is based on knowledge of the pathology of these diseases. The pathology of the periodontal tissues is not well covered in the standard text books on dental pathology. A great deal of factual material in histopathology essential to periodontia and discovered by Gottlieb, Orban, Kronfeld, Box and others is not included in the most used text books in dental pathology. Since this material is essentially pathology, it should be taught in the course of oral pathology and not left entirely for the courses in periodontia. The same is true in oral hygiene. An immense amount of experimentation and other research has been accomplished in the last twenty years in the field of oral hygiene. Research on oral bacteriology, mouth detergents, saliva, dental calculus, diet in relation to dental disease, and endocrine relationships are part of oral hygiene. Study of these researches should be included in the course in oral hygiene. The courses in periodontia should be based on this material and such of it as is indicated should be applied but not all of it needs to be taught in the courses in periodontia. Oral diagnosis can only be taught adequately by those well versed in the principles of periodontia and the sciences on which it is based. A course in periodontia taught in a school where the teachers of oral diagnosis are uneducated in periodontia loses considerable effect. Periodontia cannot be well taught as an isolated subject. Its successful teaching depends on proper preparation for it in the preceding science subjects of oral pathology and oral hygiene, and on coöperative and informed teaching in oral diagnosis.

The courses in periodontia should start with a course of prophylaxis in the sophomore year of the dental college curriculum. This should be the introductory course to the student of operative procedures in the mouth. It is well, if there is time for it, that the actual prophylactic work in the mouth be preceded by work on manikins. In this way the use of the mouth mirror and the simple instrumentation of cleansing and polishing may be learned without too much inconvenience to the patients. By arranging this course at the beginning of the student's work in the mouth, the proper emphasis is laid on prevention and hygiene. Not only is he interested at the outset in this phase of dental practice but he also gets practical information in order to better understand the reasons for procedures in operative dentistry which are aimed to avoid irritation and disease. The course in clinical prophylaxis should be accompanied by a series of lectures on the home care of the mouth and prophylactic technic.

The time required for the sophomore course in prophylaxis as estimated by the Curriculum Survey Committee of the American Association of Dental Schools is sixteen hours of class and laboratory time plus sixteen hours of clinical time. This is in accord with the practice of several of the schools where prophylaxis and periodontia get deserved recognition and apparently is sufficient for the purpose. Another thirty-two hours during the junior and senior years together, should be devoted to prophylactic work in order to keep this phase of dentistry ever before the student.

The clinical work in periodontia proper should start in the junior year. It is a continuation and extension of the course in prophylaxis. There is no great value at this stage in manikin technic. The student has already been trained in removing superficial subgingival deposits with prophylactic instruments and easily utilizes the finer instruments and technics of root surface curettage. A course of thirty-two hours in clinical work including lecture demonstrations on philosophy of treatment, charting, instrumentation, care of instruments, special home care methods, ligating and splinting, grinding for traumatic occlusion, treatment of sensitive dentin, treatment of Vincent's infection, etc., is sufficient for the year. It is practicable to have the dental class divided into sections of eight to twenty for this work with one instructor for each eight or ten students. Cases should be carefully selected in the examination clinic in order that each student gets one or more cases of well developed periodontoclasia to treat.

The clinical course in the senior year should be similar in administration and time to that of the junior. The group lecture demonstrations may be continued and devoted to methods of treatment other than root surface curettage, to dental occlusion, and other mechanical relationships affecting the periodontal tissues.

In an undergraduate course of study it is usually possible to teach one procedure only for a given end. A variety of procedures from which a dentist may choose in a given case must necessarily wait upon postgraduate instruction and practice. In the case of periodontia, one procedure is taught the students—the root surface curettage technic followed by home care instructions to keep the teeth clean, the affected tissues massaged, together with treatment of defective occlusion and other mechanical sources of irritation.

In addition to the clinical work a science course from ten to twenty hours should be offered in periodontology during the senior year. This course should consider the basic theory for the technical procedures of periodontia. It should come in the senior year rather than in prior years. The student grasps the significance of the material presented better if he has had actual experience in studying and treating periodontoclasia. It works better in practice for the student to learn procedures first and then study the theory after considerable work in the field. The science course in periodontology should cover the subjects of history,

histopathology, etiology in its biological, bio-chemical and mechanical forms, symptomatology, diagnosis, treatment, and prevention, with special emphasis on Vincent's infection. The surgical resection, coagulation resection, pocket packing and other methods of treatment are not taught except through lecture and demonstration. In the limited time allowed by a crowded curriculum it is not possible or practical to attempt to train students in all technics by which the gingivae and periodontal tissues may be successfully treated. They are taught the simplest method of successfully treating the vast majority of moderately progressed periodontia cases.

The foregoing plan of teaching periodontia will, of course, be modified to accord with the conditions in the various schools. There is no hard and fast rule by which dentistry or any dental subjects must be taught. In at least two schools periodontia is taught by the department of operative dentistry, but gets its full share of attention. The objective is to see that it is taught, that sufficient time is devoted to it, that the teachers of it are themselves competent and interested in it and that the teaching of other branches of dentistry is coördinated with it. The day should have gone by when so vital a field as periodontia in dentistry may be overlooked in the curriculum of the dental school just because State Boards of Dental Examiners do not examine particularly in the subject.

630 West 168th St.

Invitation—

THE AMERICAN ACADEMY *of* PERIODONTOLOGY invites the members of the AMERICAN DENTAL ASSOCIATION to attend the clinics and scientific sessions of its annual meetings.

Since the satisfaction and the permanence of success in operative dentistry depend to a large degree upon health in the supporting tissues of the teeth, it is desirable that dentists generally be familiar with present-day knowledge of prevention, diagnosis, and treatment of periodontal lesions.

Plan to attend the next annual meeting of the A. A. P., Atlantic City, July, 1937.

ABSTRACTS AND EXTRACTS OF CURRENT DENTAL LITERATURE FOR 1935

Periodontal Histo-pathology, Periodontal Bacteriology, Other Laboratory Researches, Clinical Etiology, Symptomatology and Diagnosis, Treatment, Home Care and Prophylaxis, Vincent's Infection, and Miscellaneous Subjects

(a) Periodontal Histo-pathology

(Continued from July, 1935 issue, page 68)

168—Barroso, R. and Vera, C.: **Für Histologie und Histogenise der Gaumenleisten des Menschen und einiger Säugetiere.** Histology and Histogenesis of the rugae in man and several mammals, in: Paradentium, 4(7): 61-74. 1935.

The authors describe the histology of the rugae as seen in man, pigs, rats, cats, and sheep. The article is illustrated with photomicrographs showing various stages in histogenesis as seen in different species of foeti.

169—Bödecker, Charles F., D. D. S. and William Lefkowitz, D. D. S.: **Gingival reattachment to enamel.** Abst. in: J. Dent. Research 15: 210-211. June, 1935.

Gottlieb showed that gingival tissue is firmly attached to enamel under normal conditions. This has been substantiated by Orban, Kronfeld, Becks, Box, Applebaum, Bödecker, and others. Attention has also been called to dangers of mutilation of this attachment by operative procedures. Experimental work on dogs, herein described, indicates that such dangers may not be real, and also shows remarkable recuperative powers of periodontal tissues. Six teeth in five dogs were removed, canals filled, replaced and held in position by splints; two of these teeth were carefully scraped free from all gingival tissue. Despite this rigorous procedure, interdental gingival papillae became reattached to *enamel* by means of Gottlieb cuticle. These findings may apply also to man, the histological structure of involved tissues being similar in both species. The only differences seem to be the form of the teeth, and the possible germicidal property of dog saliva. Therefore, reattachment of gingivae to enamel probably occurs in man *in the absence of inflammatory conditions.* (To be published in D. Cosmos; report of Research Committee, Dental Society, State of New York, 1935.)

—*Copy.*

170—Brashear, Alton D., M. S., D. D. S.: **Observations on relative sizes of nerve fibers of pulp and peridental tissues and their probable significance.** Abstr. in: J. Dent. Research, 15(3&4): 157. 1935.

Histological analysis of maxillary and inferior alveolar branches of the trigeminal nerve (cat) reveals that 44 percent of the nerve fibers are unmyelinated or small myelinated fibers less than 6 microns in diameter; 29 percent vary from 6 to 10 microns in diameter; and 27 percent are larger than 10 microns. An analysis of the stem nerve-fibers entering pulps of cat teeth show that 64 per cent of the fibers are unmyelinated, or small myelinated nerve-fibers less than 6 microns in diamenter. The rest of the fibers vary from 6 to 10 microns in diameter. In histological sections of peridental tissues, nerve fibers of all sizes occur, 20 to 25 per cent being larger than 10 microns in diameter. A review of the literature on sensory conduction elsewhere in the body produces evidence that touch is conducted by large myelinated sensory nerve-fibers; temperature, by those of a medium size; pain, by fibers of a fine caliber, both myelinated and unmyelinated. The author suggests that pain is the predominant, if not

the only, sensation elicited from the tooth pulp; and that the peridental tissue, through its supply of nerve fibers of all sizes, becomes the organ of touch for the tooth, as well as responding to other sensations.

—*Copy.*

171—Coolidge, E. D., M. S., D. D. S.: **Evidence of excessive occlusal stress in periodontal tissues of human teeth.** Abst. in: J. Dent. Research, 15: 159-160. June, 1935.

"Material from seven human jaws, and articulated plaster casts and radiograms of the teeth, were studied. The ages of the individuals at the time of death varied from 11-55 yrs. The fatal diseases may have reduced resistance and recuperative power of the entire cellular mechanisms, but different degrees of functional stress on periodontal tissue would have results similar to those found for any individual. Similar conditions have been produced artificially in animals in good health. *Teeth in good alignment and occlusal relation,* and showing normal function, had uniform periodontal spaces, rather thin cementum, and strong, well-defined periodontal fibers. Alveolar bone was well braced with trabeculae of supporting bone. *One bicuspid* in good position and occlusal relation, supporting an artificial crown to which an unsupported pontic was attached, did not reveal any evidence of trauma in the periodontal tissue, although it might have been present as a result of the extra load carried by the roots; no evidence of excessive occlusal stress was found around any of the teeth in this jaw. *Teeth abraded by occlusal wear,* but without antagonists at the time of death, had greatly thickened cementum and very narrow periodontal spaces filled with indefinite connective tissue showing very few principal fiber bundles. Alveolar bone was thin and poorly supported by bone trabeculae. Marrow spaces were greatly enlarged and fused in many areas. These findings correspond to those reported by Gottlieb and Orban, Kronfeld, Box and others. Teeth upon which the occlusal forces were greater than the capacity of the tissue to support the load were surrounded by evidences of hemorrhage, necrosis, and bone resorption on the side of the pressure, and a widened periodontal space. Several cases of cementum fracture, and many areas of necrosis were found. The most interesting case was that of a child of 11, who died of scarlet fever. The periodontal tissue of each deciduous molar, and of the four first permanent molars, had areas of necrosis in the pressure regions. These findings suggest that age is not a factor in the development of injury from excessive or abnormal occlusal stress; tissue resistance, and the recuperative power of the cells are the greatest factors."

—*Copy.*

172—Ellis, David, D. D. Sc.: **Microscopical pathology of pyorrhea alveolaris,** in: Australian J. Dent. 39: 243-247. 1935. (Synopsis of a thesis presented for the D. D. Sc. degree.)

Conclusions:

"1. Pyorrhea is not a disease entity but is the natural result of a simple local inflammation of the gingiva. While admitting that a constitutional factor may leave the tissue concerned below par the results of this study do not indicate that this is a necessary corollary.

2. Pocket formation is established by chronic epithelial proliferation with degeneration of the superficial layers of epithelium cells at the gum margin and a splitting of the epithelium at the base of the ulcer bordering the cementum.

3. The epithelial downgrowths which take place along the cementum are definitely bound by that tissue to the tooth. Cementum acts as a specialized connective tissue.

4. Epithelial remnants on the erupted cemental walls of the teeth are the nuclei upon which tartar formation occurs.

5. There is no necrosis of bone. Sequestra are not formed. Resorption of bone is progressive, beginning at the alveolar edge and not in the body of the bone. Bone undergoes a slow progressive change, firstly to osteoid and then to fibrous tissue.

6. A simple hyperemia without metabolites is not sufficient to cause marked change in the alveolar bone.

7. When a gingivitis is established, mouth bacteria enter the tissues and act as sustaining causes. This is largely dependent upon the predisposing conditions prevailing.

8. In advanced stages of the disease there is a mass microbic infection of types of organisms not yet identified and a mild osteitis is developed in the alveolar bone.

—*G. H.*

173—Kronfeld, R., M. D., D. D. S.: **The condition of alveolar bone underlying pyorrhea pockets,** in: J. Periodont. 6(1): 22-29. 1935.

A study of cross sections of pyorrhea pockets from human jaws obtained fresh from autopsy revealed the following pathological changes: The pockets were from 5-8 mm. in depth. The pocket epithelium was ulcerated and broken up into small fragments. Beneath this there was a layer of connective tissue of about 1-1.5 mm. in thickness. This was infiltrated with round cells especially immediately beneath the epithelium. The contiguous bone was in the process of resorption, histologically evidenced by the numerous osteoclasts in Howships lacunae. This process extended to slightly deeper marrow spaces too. The bone itself was normal in appearance, i.e. there was no process of decalcification. Radiographically the condition of the bone showed a lowering of the alveolar crest and an increased radioluscency.

—*S. H. S.*

174—Retterer, E.: **De la gencive et de la membrane circumradiculaire a l'état physiologiques et dans quelques conditions pathologiques.** The gums and circumradicular membrane in physiologic and pathologic conditions, in: La Semaine Dentaire, 17: 683-696. 1935.

Observation of the normal evolution of the buccal mucosa and experimentation have convinced Dr. Retterer that the gums do not arise from separate ectodermal and mesodermal origin but that both in the embryo and in the adult the epithelial cells are transformed in the depths of the epithelial lining into connective tissue elements. The tooth itself arises from the epithelial bud in the chorionic tissue. From this epithelial bud is formed a mesenchymatous element which forms in turn the dental pulp elaborating the enamel and dentine. Connective tissue is the second stage in epithelial evolution. Even in the adult the gingival epithelium continues to send prolongations known as Serres glands or epithelial debris into the circumradicular membrane. This membrane is therefore a mixed epithelio-connective tissue. The membrane is not a fixed one; it thins with advancing age while the cortical thickens at its expense. The interrelated growth between the epithelial debris and connective tissue is dependent on local and general conditions. There exists a physiological equilibrium between the epithelial debris and connective tissue. When the systemic resistance is lowered there is an absence of mechanical action. A pathological state arises wherein there is an appearance of cells with a clear cytoplasm in the gingival epithelium. Likewise in pyorrhea there is an appearance of white cells which destroy the epithelium and cause the characteristic destruction of tissue. This condition is an epithelial degeneration caused by the absence of masticatory action and is augmented by nutritional and systemic disturbance. This epithelial degeneration occurs likewise in the circumradicular membrane. Micro-organisms penetrate the epithelial lining now deprived of its intact cells and attack the epithelial debris in the membrane. Formation of new connective tissue ceases and an intense inflammatory reaction occurs.

The regressive power of the epithelial islands and cells of the interdental gingiva follow in this order: 1) Production of a syncytial cytoplasm; 2) Resorption of the cytoplasm; 3) Freedom of the cells as white corpuscles.

The evolution and vitality of the gingival epithelium depends on the local factor of mechanical action, i.e. mastication and is modified by the systemic resistance which controls the tissue vitality. A high level of general health and sufficient masticatory action is the best means of avoiding gingivitis and pyorrhea.

Morphological evidence shows that carnivorous animals possess a type of tooth fitted for their masticatory purposes. This manifests the extreme importance of mechanical action on the evolution of teeth. The integrity of the teeth and surrounding tissues depends on the general health and the mechanical factor.

—*G. H.*

175—Rosebury and Seigel: **The work of Schour and his collaborators on the dental influence of the parathyroid and pituitary glands.** Review, in: J. N. Y. A. D., 2: 108-115. 1935.

Schour and his co-workers have undertaken a complete systematic study of the relationship between the endocrine glands and the teeth and their supporting structures. In addition to their report on the response of the hard tissues of the teeth to endocrine imbalances they have demonstrated soft tissue changes.

In hypophysectomized rats it was found that the epithelial attachment was normal near the gingival crest, but atrophied or absent at the cemento-enamel junction. There was no evidence of diffuse alveolar atrophy or paradentosis. The rate of tooth eruption was retarded.

—*S. H. S.*

176—Siegmund, H. and Steinhardt, G.: **Osteitis fibrosa and paradentosis,** in: Zahnärtzliche Rundschau, per L. Houpert, in: Rev. de Stomatol. Mar., 1935. Abstract, in: Dent. Record, 55(12): 735. 1935.

Becks and Askanazy-Held have observed that in experimental generalized osteitis fibrosa the jaws and alveoli are affected by the disorders, and that the appearances have a considerable analogy to those seen in paradentosis in man. The authors therefore examined five cases of Recklinghausen's osteitis fibrosa and Paget's disease. By comparing the results with the preparations from experimental osteitis fibrosa which they and Wannenmacher had produced, they were able to show that in the jaws the processes of osteitis fibrosa and of paradentosis were very different. In osteitis fibrosa the absorption always begins in the medulla of the interdental alveolar spine and is accompanied by a marked hypercementosis. In paradentosis, on the other hand, there is no hypercementosis, and the rarefaction takes place on the part of the alveolus in contact with the root.

—*S. H. S.*

177—Skillen, W. G., D. D. S., and Lundquist, G. R., D. D. S.: **Experimental gingival injury in dogs,** in: J. Dent. Research, 15: 165-166. June, 1935.

These experiments were undertaken to determine character of repair in healthy tissues, to ascertain (a) what might be expected in diseased tissues and (b) possibility of reattachment of peridental membrane and epithelium. Types of experiments: (1) severing of gingival tissues from tooth surface to level of bone, i.e. artificial pocket formation; (2) severing of gingival tissues from tooth surface to level of bone, curettement of root surface followed by use of sodium sulphide to dissolve any remaining epithelium; (3) typical flap operation; (4) typical gingivectomy. Teeth were marked and measurements taken of position of gingival crests and depth of injury. In most instances there was only partial and sometimes very little peridental-membrane reattachment. (Resorption of tooth surface was a frequent occurrence in this region.) Denuded surfaces apparently were rapidly covered with epithelium, irregular and disorganized in structure. Thus the healed pocket was usually very deep. In only very few instances did a nearly normal restoration occur. The shallowest pocket was obtained in gingivectomy. In no instance was there any indication of epithelial reattachment to *enamal** surface. There was, however, epithelial attachment to *cementum** in some cases. Various amounts of calculus were found in most pockets.

* (Italics not in original.)

—*Copy.*

178—Wheeler, Russell: **A comparison of periodontal attachment levels, anterioposteriorly,** in: Dental Digest, 41: 261-263. 1935.

The height of the normal gingival tissue around the teeth is dependent on the height of the periodontal attachment. Injury to this attachment during dental operations is frequent and the author pleads for greater care in operating and for consideration of anatomic detail.

From his studies on extracted teeth the author finds that the periodontal membrane is attached about as high on the lower as on the upper teeth. The membrane attachments are at their highest points at the midline and decrease gradually in distal progression until the mesial of the first bicuspid is reached. From this point to the third molar curvatures are slight. It is also interesting to

note that the height of the periodontal attachment on the various teeth is almost parallel to a line drawn through the contact points.

—*G. H.*

(b) Periodontal Bacteriology

(Continued from 6(2): 70. July, 1935)

57—Beust, Theodore B., M. D., D. D. S.: **Fusiform bacillus,** Abst., in: J. D. Research, 15(3 & 4): 167. 1935.

Descriptions of these oral inhabitants have been based on inadequately stained preparations. As a result, confusion exists concerning their morphology, cytoplasmic contents, and reaction to reagents. Photomicrographs offered to support claims of successful culture bear no resemblance to the organisms that occur in the mouth. Studies of forms, flagellation, metachromism, tinctorial behavior, and developmental history, indicate that Bac. fusiformis associated with Vincent's infection has not yet been artificially cultivated.

—*Copy.*

58—Cahn, L. R., D. D. S.: **The penetration of microörganisms into the gum as a cause of chronic and recurrent gingivitis,** in: D. Cosmos, 77(3): 264-268. 1935.

With the help of a series of photomicrographs, the presence of microörganisms in the tissues in cases of gingivitis is shown. There is normally an abundant flora of microörganisms in the mouth. These are in the main innocuous. When the tissue resistance is lowered they penetrate and, with the change in environment, become pathogenic. An intact stratum corneum is the best defense against invasion.

It is the lingering presence of the microörganisms within the tissues that is the cause of chronic and recurrent gingivitis, he believes.

The author treats briefly on methods of destroying bacteria and fungi within the tissues. He finds the home use of a salve of ammoniated mercury used as an inunction, very efficacious. Correction of predisposing causes is of course paramount.

—*S. H. S.*

59—Okell, C. C. and Elliott, S. D.: **Bacteremia and oral sepsis with special reference to etiology of subacute endocarditis,** in: London Lancet, 2: 869-872. Oct., 1935. Abst. (Condensed), in: J. A. M. A., 105(25): 2115. Dec., 1935.

When searching for source of streptococci concerned in production of subacute bacterial endocarditis, attention must inevitably be turned to oral cavity. Majority of endocarditis streptococci are of the salivary or streptococcus viridans types. These abound in the mouth and its neighborhood, especially in conditions of oral sepsis such as "pyorrhea of the gums," while elsewhere in the body they are found only in small numbers and under exceptional conditions. In persons with severely septic mouths, non-haemolitic streptococci (mostly of the viridans type) may enter the bloodstream even in the absence of any obvious trauma. The authors have not yet determined whether minor traumas such as brushing the gums or chewing have any effect on the occurrence of bacteremia. A "leak" of bacteria into the bloodstream which may be of little or no consequence in a well, normal person might determine the infection of diseased or malformed valves. The direct observation of such an invasion of the blood provides a tentative explanation of how streptococci reach the heart valves in cases of subacute bacterial endocarditis.

60—Rosenow, E. C., M. D.: **Elective localization and cataphoretic velocity of streptococci isolated from pulpless teeth and other foci of infection.** Summary of results, in: J. N. Y. Acad. Dent., 2: 92-98. 1935.

Pulpless teeth whether X-ray negative or positive, especially among persons having systemic disease, are nearly always infected with alpha streptococci that are highly sensitive in primary culture to oxygen and other requirements of growth. Aerobic cultivation of streptococci on blood agar and in ordinary broth usually destroys the property on which elective localization depends. Elective localization is maintained for a longer time when organisms are kept in dextrose brain broth or soft dextrose brain agar. Electively localizing streptococci were isolated consistently from the depths of

pyorrhea pockets, enamel organs of unerupted teeth, from the apical ends of aseptically removed pulpless teeth and from trephined apical ends of pulpless teeth successfully treated for bone rarefaction.

Streptococci isolated from pulpless teeth, depths of pyorrhea pockets, alveolar abscesses, dental sinuses, tonsils, etc., tend to produce lesions electively, in the tissues of inoculated animals corresponding to those affected in the original person. The clinical improvement following elimination of infection in dental foci and the exacerbation of symptoms of the systemic disease following removal or attempted elimination of foci leaves little doubt of the etiologic importance of symptom-producing, and symptom-less, localized infections in dental and other regions.

Streptococci isolated from cases of chronic infectious arthritis have a high arthrotropic cataphoretic velocity. Streptococci isolated from cases of chronic encephalitis have a neurotropic cataphoretic velocity much lower in value. Dosage by short wave lengths of electricity caused arthrotropic streptococci to lose affinity for joints and acquire affinity for the brain. The opposite could be done with neurotropic streptococci.

The shift in the distribution curves of the cataphoretic velocity of streptococci isolated from infected teeth of well persons and of those suffering from chronic arthritis and other chronic diseases during epidemics of influenza in the winter and spring indicates that the increased incidence of acute pulpitis and of acute infections of pulpless teeth during these months may be attributable to the increase or a change in the virulence of streptococci.

Cataphoretic velocity or electrical charge is a determining factor of systemic localization of streptococci. The marked specificity of the causative streptococci indicates that active and passive immunization with specific vaccines and serums are necessary in the treatment of diseases attributable to streptococci.

—G. H.

(c) Other Laboratory Researches

(Continued from 6(2): 72. July, 1935)

66—Bibby, B. G.: Formation of salivary calculi, in: D. Cosmos, 77: 668-675. 1935.

The various theories on the formation of salivary calculus are reviewed. Although little unanimity of opinion is present among workers in the field, the prevailing views fall into two groups; first, that physicochemical agencies precipitate Ca salts from the saliva; second, that bacteria are the causative factors in the precipitation. Hunter, Burchard, and Kirk believe that precipitation results from the loss of CO^2 from the saliva and the action of basic substances such as ammonia formed in the mouth. Prinz claims that a surface concentration of salivary colloids sets free Ca salts which are carried out of solution by the sinking of the colloid pellicle. These theories, however, do not explain the formation of calculus in certain localities only, in many mouths. Smith's phosphatase theory likewise fails to explain the localization of calculus in certain regions, as does the bacterial theory.

Experiments were performed to determine the following problems:

1) Effect of bacteria and the loss of carbon dioxide upon the precipitation of Ca salts from saliva.
2) Effect of alkalies on the precipitation of Ca salts from saliva.
3) Comparative effects of phosphatase, bacterial activity and the loss of carbon dioxide on precipitation of Ca salts from saliva.
4) The effect of oxygen tensions on the precipitation of Ca salts from saliva.
5) Effect of bacterial cultures on precipitation of Ca salts from unstable solutions.
6) Presence of special bacteria in calculus deposits.
7) Structure of salivary calculus.
8) Cultural investigations of bacteria in calculus.

A review of the experiments reveals that no calcareous deposits were precipitated from saliva unless bacteria were present. Bacteriological and histological studies showed that Leptotrichia (rather than as Naeslund claims, Actinomyces) were the predominant organisms in calculus. Explanation of calculus on purely a bacterial basis does not, however, explain the localization of calculus around salivary ducts, the scarcity of bacteria in calculus around dogs teeth, and the heavy but uncalcified deposits of Leptotrichia found on many teeth.

The experiments also showed that precipitation of Ca salts from saliva may be brought about by carbon dioxide loss. However, loss of carbon dioxide must not be confused with the fixation of calcium precipitates into calculus deposits. It is possible that bacteria are the important factor in the fixation. No single cause has been found responsible for the formation of salivary calculus. The author suggests that a physico-chemical process leads to the initial precipitation of Ca salts while bacteria play a role in fixing these precipitates as deposits.

—*G. H.*

(d) Clinical Etiology

(Continued from 6(2): 80. July, 1935)

311—Beregoff, Pauline, M. D.: **Infection of the mouth in relation to dysentery, with report of a case of intestinal amoebiasis,** in: Can. Med. Assoc. J., 32:6. 1935.

An extensive study of various mouth infections revealed that in pyorrhea alveolaris cases amoebae were present in the gingival pockets. In fifty per cent of the cases studied the amoeba closely resembled the Entamoeba histolytica in fresh and in stained preparations.

An interesting case report is given in the article, of amoebic dysentery accompanied by sore gums and loose teeth. Fourteen injections of emetin apparently cured the condition, i.e. the amoebic dysentery.

After checking the patient's environment, the hygienic condition of the home, and finding no other member of the family similarly afflicted, the conclusion was reached that the long standing mouth infection was the predisposing factor of the intestinal condition. Smears from the infected pockets about the teeth showed practically the same picture as found in the feces.

—*G. R. S.*

312—Bergmani, M.: **Les facteurs constitutionels dans l'étiologie des maladies du paradentium.** Constitutional factors in the etiology of paradental diseases. Abst., in: Revue de Stomat., 37(10): 694. 1935.

Attention is called to the importance of individual predisposition in the causation of diseases in general.

There are three etiological factors in the predisposition to paradental diseases:

1) Constitutional disease;
2) Endocrine disturbances;
3) Heredity, age, sex.

Our knowledge on the subject is too vague to definitely establish a correct single factor. The etiology of paradental diseases will be determined only when it is viewed in its broadest aspects, from an anatomical, physiological, and pathological point of view.

—*S. H. S.*

313—Berke, J. D., D. D. S.: **Etiology and histology of dental tartars,** in: D. Cosmos, 77(2): 134-139. 1935.

The author presents a series of photomicrographs showing the structure of dental tartar. On the basis of his histological studies he comes to the following conclusions.

"(1) An organic matrix exists in all deposits found on the teeth, including the clinical stains and tartars. There was found no pure crystalline deposition of calcium salts about the teeth.

"(2) The organic matrix consisted of fungi, bacteria (mostly cocci), phagocytes, fibrinous matter, a few epithelial cells, hemaglobin and, in some instances, organic stains.

"(3) The arrangement and proportion of these components varied with the location of the tartar, i. e., coronal, cemental and inflammatory.

"(4) From clinical and experimental observations it is apparent that local factors such as the presence of microörganisms,

saliva, and at times a concurrent gingival disturbance, even though incapable of being seen clinically, are involved in the formation of calcareous deposits about the teeth. Systemic disturbances such as those of Ca-P metabolism undoubtedly influences tartar formation."

—S. *H.* S.

314—Boyens, Paul J., D. D. S.: Etiologic factors in various types of paradentosis, in: D. Cosmos, 77(1): 1-11. Jan., 1935.

There are two and perhaps three fundamental pathological processes underlying paradentosis: ***inflammation*** and ***bone resorption.*** A third, the ***involution of epithelium along the surface of the root,*** is claimed by some to be independent of the inflammation and bone resorption.

There are systemic as well as local factors involved. Those systemic factors which cause ***inflammation*** are, (1) Vitamin deficiencies, especially vitamin C, (2) Endocrine disturbances. *Systemic* conditions that may influence ***bone resorption*** are, (1) Lack of sufficient intake, (2) Excessive demands for calcium, as in pregnancy and lactation, or too rapid growth, (3) Improper digestion of calcium bearing food, as may occur when the bile and pancreatic juice is deficient, (4) Failure in conveyance of the calcium to the bone which occurs in rickets, (5) Inability of the bone to utilize the minerals from the blood because the organic matrices are not being built for its retention or because they are degenerating, (6) Reversed bone metabolism, wherein the blood robs the bone of its calcium, as in cases where hypothyroidism is associated with hyperparathyroidism. Other less specific *systemic* conditions predisposing to bone resorption are, (1) neuroses, (2) foci of infection, (3) salivary calculus associated with calcium imbalance, (4) lowered vitality.

We have certain systemic factors which strongly indicate a *specific* effect upon the paradentium, as for example the gingivitis of pregnancy, and the affinity which metals have for the gingival tissue.

The *local* causes are divided into three main groups. (1) Tissue irritants causing inflammation, (2) Pressure causing bone resorption, (3) Lack of tone and lack of development and through hypofunction. Tissue irritants are subdivided into (a) bacterial, (b) chemical, (c) mechanical.

A chart is included which classifies paradental diseases into seven types according to the system adopted at the Eighth International Dental Congress (Paris, 1931).

—S. *H.* S.

315—Broderick, F. W.: The mouth as an autobiography, in: Med. Rec., 141: 285-287. Mar., 1935.

The article is intended to show the importance of the mouth as an index to the general metabolism, past, present and future.

Broderick has long been an advocate of the theory of systemic origin of periodontoclasia. In this article he takes up a suggestion first advanced by Pemberton, that caries is associated with a sympatheticotonia, and that periodontoclasia is associated with a vagotonia. (See Broderick's "Dental Medicine," for complete explanation of his theories. Book reviewed, 7(1). Jan., 1936.)

—S. *H.* S.

316—Brudevold, B.: A statistical survey on paradentosis, in: Norske Tannlaegeforenings Tid., 7: 1935. Abst., in: Zahnärtz. Rundsch., 44: 2131-2132. 1935.

The author hoped to determine how frequently the predisposing factors of paradentosis as malocclusion and concretions were present in 366 cases. Gingivitis was also considered as it frequently leads to paradentosis.

The findings were connected up with frequency and treatment of dental caries.

Conclusions:

1) In case a socialized program of the care of the mouth was instituted, a symptomatic handling similar to that for the school child now in use would be worthless since the caries frequency is too great. The foundation should be extensive with thorough prophylaxis, and better hygiene, and then symptomatic treatment.

2) At the age of 21 years, 50 per cent of the cases were already suffering from some gingivitis and needed a prophylaxis to ward off future paradentosis.

3) Half of the community needed orthodontics to correct malocclusions. The error that mouth care consists of stopping cavities only should be corrected from the standpoint of the public view. Prophylaxis should be stepped up to first importance.

—*N. K. B.*

317—Ellis, D.: **There is no bone necrosis in pyorrhea.** Excerpt, in: Dent. Mag. and Oral Topics, 52(12): 1184-85. 1935.

The claim is made that:

1. Pyorrhea is not a disease entity—but an end result of local gingivitis;

2. Pocket formation is established by chronic epithelial proliferation; with degeneration of top layers at gum margins and splitting at the base;

3. Epithelial downgrowths along the cementum are definitely bound by that tissue to the tooth;

4. There is no necrosis of bone. Resorption of bone is progressive beginning at the alveolar ridge and not in the body of the bone. The change is gradual, first to osteoid, then to fibrous tissue;

5. Once the gingivitis is established, bacteria enter and act as sustaining causes;

6. In advanced cases there is infection, and a mild osteitis is produced in the alveolar bone.

—*S. H. S.*

318—Finni, J., and Gottlieb, J. S.: **A report on excessive dental calculi formation,** in: D. Cosmos, 77: 1173-1176. 1935.

The authors present the case of a woman hospitalized for psychiatrical reasons. Physical examination revealed only an inactive tuberculosis. Mouth hygiene was poor with but little salivary calculus and extensive pyorrhea. About four years after hospitalization (at which time all the upper teeth were removed) massive deposits of hard greyish white calculus were noted almost completely covering the lower teeth in some instances extending to the apices of the teeth. There was no tenderness or bleeding of the gingiva.

The case is important because it presents evidence useful in eliminating possible causative factors of calculus formation. Oral infection, lack of hygiene as well as lack of mastication can not be regarded as primary etiologic factors since they antedated the calculus by several years. Nutrition during the period of calculus deposition was, however, poor. Although it is believed by some that organisms are factors in calculus deposition no calculus was deposited in the period of active infection. The rapid formation of calculus seems to be dependent on some inherent abnormality.

—*G. H.*

319—Held, Dr.: **La prosthèse dans la prophylaxie et la therapeutique des paradentoses.** Prosthesis in the prophylaxis and therapy of paradentosis, in: L'Odontologie, 73: 432-442. 1935.

Clinical and experimental research has shown that paradentosis is to be considered as the expression of a systemic disorder. Local causes (tartar, infection, traumatic occlusion, etc.) are the aggravating factors, which, in a system weakened by some disorder, may give rise to severe periodontal lesions. The author discusses traumatic occlusion as one of such local aggravating factors. In a favorably weakened system, teeth affected by traumatic occlusion are the first to become pyorrhetic and prosthesis as a correcting factor plays an intimate part in the prevention and treatment of paradentosis. The aim of partial prosthesis is not merely to fill a space but to conserve the remaining structures in the mouth. The biological viewpoint is necessary. Prosthetic treatment is indispensable when traumatic occlusion is a determining factor of the paradentosis.

—*G. H.*

320—Jones, M. R., Ph. D.: **Our changing concept of an "adequate" diet in relation to dental disease,** in: D. Cosmos, 77: 535-549, 651-662, 747-763. 1935.

The article deals primarily with the relationship of diet and dental caries, with some side remarks on scurvy, osteomalacia, and rickets; and what constitutes an adequate diet. The author makes an effort to analyze the data reported by some of the eminent workers in the field of nutrition. The reports are too numerous to discuss in this abstract and do not deal with periodontal disease. We do wish, however,

to point out some of the author's passing references to periodontal disease in her analysis.

First, she notes a direct relationship between excessive amounts of vegetables in the diet and periodontoclasia. This is drawn from a table showing the distribution of calories among certain classes of foods composing the diet, and its potential alkalinity in relation to dental diseases, reported from various institutions and places (table 5).

A chart analyzing the potential alkalinity of diet in relation to caries and periodontoclasia reveals that in a highly alkaline diet the teeth are caries free, loosen and fall out, because of atrophy or brittleness of bone. The Samoan adult is practically caries free, yet 98 per cent of Samoan adults over 35 years of age have periodontoclasia. Ferguson ascribes this to lack of hygiene and tartar accumulation. The calculus may accumulate because of the high exclusive vegetable diet rich in minerals and high in potential alkalinity.

—*G. H.*

321—Kaushansky, L. I.: **The etiology of so-called alveolar pyorrhea: influence of diet on mineral metabolism, experimental study,** in: D. Cosmos, 77(1): 69-74. Jan., 1935.

Many workers have found a positive correlation between alveolar pyorrhea and mineral metabolism disturbance in a large majority of cases. Citron found that 96.5 per cent of paradental cases show hypercalcemia.

The paper represents experimental work on the problem as to whether and how much acid and alkaline diets influence the concentration and ratio of electrolytes in blood serum; also whether it is possible to effect the mineral composition of the solid tissues of the teeth by varying the diet. The analysis was for K, Na, Ca and Mg changes.

Four grown up dogs were used; two were fed acid rations, two were fed an alkaline diet.

Blood analysis of the alkaline-eaters showed—(1) Ca content considerably reduced from that during the normal diet. (2) The K/Ca ratio was above normal. (3) The Mg was increased.

The "acid-eaters" showed (1) decrease in the calcium content below that of the normal diet, but not as great as in the alkaline group. The K/Ca ratio decreased. The Loeb ionic coefficient changed with both pairs. Mg fluctuated in inverse proportion to calcium content, i. e., where Ca content was high, Mg was low and *vice-versa.*

Analysis of enamel showed a greater increase in Ca content over normal in "acid-fed" dogs than in "alkaline-fed" dogs. Mg and Na content was changed but not uniformly. The K content was decreased with both diets.

The Ca, K, Na and Mg changes in dentine were not in any one direction, i. e., the content either increased or decreased with both pairs.

—*S. H. S.*

322—Leonard, H. J., D. D. S.: **The diagnosis of conditions leading to destruction of the teeth and investing tissues,** in: D. Cosmos, 77(3): 276-281. 1935.

The different theories regarding the causes which lead to tooth loss and their diagnosis are presented.

Systemic factors play an etiologic role, only in that they aggravate the local conditions. Among systemic causes of gingivitis and periodontoclasia is overeating, with consequent deposition of materies alba and calculus on tooth surfaces. Dietary deficiency and endocrine disturbances are also contributory agents.

The local causes are divided into three groups: Infection, mechanical trauma, and insufficient functional stimulation. These can be detected even before the onset of the pathologic process. There are certain conditions in the mouth which predispose to gingivitis and periodontoclasia, viz.: overhanging fillings and crowns, rough enamel, food impaction due to loss of contact either through caries, drifting, or loss of teeth. Such conditions should not exist long, but should be corrected by the attending dentist.

—*S. H. S.*

323—Lewis, A. B., D. M. D.: **Oral manifestations of endocrine disturbances, in myxedema,** in: D. Cosmos, 77: 47-49. 1935.

Whenever the cause of periodontoclasia is obscure and local factors are not evident, suspicion of a systemic disturbance should be raised. The author presents a case of

pyorrhea resulting from myxedema (hypothyroidism). Mobility of the teeth appeared five years before the acute symptoms of the endocrine disease. At this time oral investigation revealed no calculus formation and little exudate from the pockets. Systemic treatment of thyroid and iron extracts resulted in a tightening of the teeth and improvement of the general symptoms. X-ray revealed an increased density of the alveolar bone. No local treatment was instituted. More importance should be attached to systemic factors in the diagnosis of periodontal disease.

—*G. H.*

324—Lüben, Hans: **Can an inflammation of the gums be produced by an absorption of lead?** in: Deutsche Zahnärtz. Wochensch., 38: 1125-1126. Nov., 1935.

Lead poisoning in industry occurs almost as frequently as the diseases from inhaling dust seen in the occupation of mining. They are both at the top in the list of occupational diseases.

The lead reaches the body in three channels: the skin, the digestive tract, and the respiratory tract. The most frequent is the respiratory tract, by inhalation. The toxic effects of lead dust can be produced by small amounts very easily, due to the large absorption surface of the lungs. All compounds of lead whether soluble or insoluble are absorbed from the epithelium of the lung. The lead therefore goes directly into the blood stream as lead albuminate. This is deposited mostly in the bones and becomes injurious when this becomes free and travels in the blood stream.

The first signs of lead poisoning are a lowering of the total red blood corpuscles and the blue line on the gums. This blue line consists of lead particles which are deposited in the capillaries of the mucous membrane of the gingiva. The hydrogen sulfide in the food changes the lead albuminate into lead sufide (insoluble) and shows up as a fine line on the gingival margin. This is also seen in the cheek mucous membrane.

The author examined 5,000 reports of cases handed in to the Industrial Hygienic Institute of Münster 1925-1934, to determine whether the presence of a gingivitis, loosening of the teeth and the presence of calculus was a test of lead poisoning. In the 5,000 cases, 862 were suspected of lead poisoning and 357 cases in which the signs of suspected poisoning were not present, but nevertheless were included in the report.

In the cases of poisoning, special attention was given to the condition of the gums. This was true also of the suspected group. These cases amounted to 1219 reports. Finally, after examinations of mouths, it was found that there is no relationship between lead poisoning and gingivitis.

Conclusions: 1) Gingivitis is not more frequent in actual lead poisonings than in suspected cases in which it was not conclusively established.

2) In industrial diseases, gingivitis is seen whether the cause is lead poisoning or any other cause.

3) There is no relationship between lead poisoning and gingivitis.

4) In gingivitis, a blue line of lead poisoning can occur early, just as it can in healthy gingiva.

—*N. K. B.*

325—Luque, M. A.: **Etiologica de la poliartritis alveolodentaria.** Etiology of paradentosis, in: Colombia Odontologica, 1: 7-26. 1935. Abst., in: D. Cosmos, 77: 916. 1935.

In this paper only the etiology of paradentosis is studied. The theory of hypercalcification of the hard tissues and retrogression of the pulp is sustained. A comparison or parallel of the antagonizing causes of dental caries process and of parodontic states is made. In paradentosis a vasomotor disturbance represented by dental hypercalcification of the hard tissues, with hypocalcification of the alveolar structures accompanied by blood alkalosis is present; while in caries there is a hypocalcification of the hard tooth structures and hypercalcification of the bony alveolar structures with acidosis.

A brief history of paradentosis beginning with Fauchard's description and following mostly the French school and authors is given. With regard to tartar deposits the views of Riggs, Younger, Bödecker and Pierce are discussed. The infectious theory is discarded following Imbert's argumentations, as is also the hereditary factor.

With regard to occlusal trauma as a pathogenic factor, the author states that the worst cases he has observed are those in which the occlusion is normal, although it can not be denied that, owing to metabolic disturbances it must be considered a secondary factor.

The arthritic theory is discarded after studying the work of the different authors who have contributed to its explanation especially that of Endelman.

Speaking of the metabolic functions of the pulp, this organ plays a very important role in the calcic equilibrium and metabolism of the hard tissues, while endocrine disturbances of its functions of mineralizing or demineralizing the tooth structures incline the balance to one side or the other, i. e. paradentosis or caries.

Luque's conclusions are that paradentosis is a disturbance of the vegetative system, dental hypercalcification with alveolar calcic resorption; that it is not infectious; that it is not necessarily a suppurative lesion; is more frequently found in aged persons due to overmineralization of the dental structures; it has a general systemic pathogenesis, so local treatments are practically useless.

Caries is due to sympathetic nervous disturbances.

—*G. H.*

326—Merritt, A. H., D. D. S.: Common diseases of the gums and their treatment, in: D. J. Australia, 7: 76-82. Feb., 1935.

Merritt presents a brief discussion of gingivitis, Vincent's infection and periodontoclasia in which he presents the pathology, etiology and treatment of those diseases. He divides the etiologic factors in periodontoclasia into local and systemic. The local factors, common to all cases, and without which pyorrhea could not occur are two in number; namely, lowered resistance of periodontal tissues and bacterial invasion. The systemic factors probably deal with an acid-base balance disturbance due to dietetic or nutritional abnormalities. Under treatment, we eliminate the disease by operative procedures, namely curettage, gum resection and by correcting the nutritional abnormality. The prevention and treatment of periodontal disease is a highly important phase of dental practice.

—*G. H.*

327—Mestral, Dr.: Mastication et paradentose. Mastication and paradentosis, in: Semaine Dentaire, 17. 1345-1350. 1935.

Among the local causes of paradentosis that of improper mastication is of prime importance. The masticatory pressure has a double effect on the paradentium: 1) it favors the venous depletion of the perialveolar space, 2) it stimulates the alveolar process through the medium of the peridental ligament. Intra-alveolar resistance and circulation depend on light occlusal trauma.

It is well known in medicine that physical irritation will cause osteogenesis. Likewise pressure on the teeth and paradentium through mastication will remove venous stasis with its consequent increase in carbon dioxide and bone resorption, and will increase osteogenetic power by means of physical stimulation.

Mastication by its control of the salivary function may have a profound effect on the gastro-hepatic function. Besides the Ca and mucin contents of the saliva, which themselves have a possible effect on gastric digestion, there is a possibility that intestinal toxins and hormones are present in the saliva. The pyloric function, moreover, is more or less dependent upon the acidity of the gastric juice. The alkalinity of the saliva may, therefore, play a role in pyloric function. Pyloric dysfunction may result in a functional surcharge of the liver allowing certain toxic protein products to pass through, thus favoring auto-intoxication or arthritis, conditions predisposing to paradentosis.

The reëstablishment of a normal mastication will fortify the paradentium, will relieve hyperacidity, and favor proper hepatic function.

—*G. H.*

327a—Paradentoses among the Abyssinians, in: British Dent. J., p. 626. Dec., 1935.

This work is a good case against Hruska's theory that dental disease is hereditary. The tribes of Abyssinia are of Hamitic

origin but live at varying altitudes; surprisingly, their dental conditions are markedly different.

A nomad group with a sparse diet of goat's milk, meal mash, no vegetables, no vitamins C, D or E, no cereals, but plenty of sunlight. At 40 years they are mostly edentulous. They use twig-points to rub their teeth clean; food is packed down into the crevices. Periodontoclasia and loss of teeth follow early. Children of eight years show heavy tartar, soft and yellow. Tatooing of gums is widespread. Caries is rare, and when present is of the rapid kind.

The same race, when adopting civilized life, with carbohydrates and sweets in their diet, shows rampant caries. The tartar formed is dark and difficult to remove.

Europeans show the worst paradentoses. Change of altitude with changes in blood pressure are also factors in altering oral conditions. Paradentoses are rare at very low levels.

328—Rawengel, G.: **Für Kasuistik der Paradentosen.** Casuistry in Paradentosis, in: Zahnärtz. Rundsch., 44: 27-30. 1935.

The author endeavors to show that psychic factors play a part in the etiology of paradentosis. He discusses four cases out of his practice.

—*N. K. B.*

329—Reznik, L.: **L'autovaccination dans le traitement des enteropathies microbiennes, accompagnées de lésions inflammatoires de la region gingivodentaire** (Thèse de Paris, 1935). The use of auto-vaccine in the treatment of intestinal microbic invasion accompanying inflammatory lesions of the gingiva. Review, in: Revue de Stomat., 37(11): 775. 1935.

Chronic microbic intestinal diseases are frequently accompanied by inflammatory lesions of the gingivae, especially in those patients in whom the anatomical structure favors blood stasis at the gingival margins. According to René Vincent this is, in effect, an entero-gingival syndrome. Vincent further states that the presence of intestinal microörganisms can be demonstrated in gingival blood cultures. Thus, specific autogenous vaccination gives "interesting results" in general, as well as in local therapy.

—*S. H. S.*

330—Ribble, R. D., D. D. S.: **Pyorrhea at age of twelve,** in: D. Cosmos, 77 (11): 1135. 1935 Correspondence.

The author describes a case of periodontoclasia beginning at the age of twelve. The following etiological factors were observed: (1) traumatic occlusion; (2) malocclusion; (3) non-occlusion; (4) salivary calculus; (5) serumal calculus; (6) debris; (7) food impaction; (8) ignorance of mouth hygiene.

The secondary factors were: (1) disturbed calcium metabolism; (2) heredity -a) syphilitic father, b) alcoholic mother; (3) decomposition of food particles.

Systemic investigation revealed a disturbed calcium metabolism.

—*S. H. S.*

331—Richards, S. A., D. D. S.: **Observations on the cause of pyorrhea alveolaris** (paradentosis), in: Dent. Mag. and Oral Topics, 52(7): 635-640. 1935.

Muscular action of the tongue, lips and cheeks is the direct cause of pyorrhea. This acts by compressing food debris and other waste products mixed with saliva between the tooth and the gum, in the form of calculus. This is called the "Direct Pocket." There is another type, the "Indirect Pocket," which is due to indirect muscular action. This is, in effect, traumatic occlusion.

The author claims that subgingival calculus is formed mainly from "food wastes" and not from the blood or saliva as is commonly believed.

—*S. H. S.*

332—Richards, S. A., D. D. S.: **Pyorrhea: A further criticism of the orthodox theory,** in: D. J. Australia, 7: 442-445. 1935.

The author is of the opinion that the actual cause of paradentosis is entirely local. The role of systemic disease and nutritional errors is as yet disputed. They probably are responsible for predisposition and susceptibility.

There are two types of pockets, direct and indirect. The more common direct pocket occurs on lingual surfaces mostly and is evident where unclean surfaces are subjected to friction by the overlying parts, such as the tongue and muscles; which force foreign material into the subgingival

space. In the indirect or rarer form the pocket is waiting to receive debris and other material that may chance to enter. Indirect muscular action, exerted by the muscles of mastication, when transmitted to a section of the peridental membrane by a traumatized tooth results in deep atrophy and subsequent peridental detachment.

Resistance to pyorrhea is dependent on the degree of the strength of union between the soft tissues and the teeth and supporting bone. In this respect heredity plays a part. The amount of waste material on the teeth and the tissue attachment determine the severity of the disease.

—*G. H.*

333—Risser, S., D. D. S.: **The relation of diet to periodontal disease,** in: Texas D. J., 53(5): 2-9. 1935.

There are many theories concerning the etiology of periodontoclasia. Some believe that the etiologic factors are local, some believe they are systemic, and others believe it is a combination of the two. The author presents some information concerning the possible relation of diet to periodontoclasia. She brings out the necessity for a proper supply of minerals, vitamins, carbohydrates, proteins and fats. The results of each vitamin deficiency and its possible relationship to the teeth and investing soft tissues are discussed. Dr. Risser stresses the importance of sufficient ingestion of calcium and phosphorous, the average daily requirement being 20 grains of the former and 33 grains of the latter.

To meet the daily requirements for good nutrition, a standard basic diet is presented, as used by the Washington Nutritional Research Foundation.

—*S. H. S.*

334—Roy, M., Dr.: **Ostéomyélite grave du maxillaire superieur consécutive a une sinusite pyorrhéique.** Severe osteomyelitis of the maxilla following a pyorrhetic sinusitis, in: L'Odontologie, 73(12): 822-828. 1935.

Dr. Maurice Roy presents a case taken from his records in which the patient complained of symptoms in the upper right side of the maxilla. There was a copious discharge of pus from this region, accompanied by a flow of pus from the nose. The upper right 2nd bicuspid and first molar were removed but the condition grew worse. There was an oral opening into the suppurative maxillary sinus. This was later followed by a severe osteomyelitis and an extension into the temporal fossa, through the zygomatic fossa. The remaining teeth on the upper right side anterior to the second bicuspid space were loose and partially surrounded by necrotic bone. These were removed. The condition was cleared up.

The author was at a loss to determine the etiology of this condition.

Some time later the same patient returned with a similar condition on the left side of the superior maxilla. The symptoms and progress were the same, including the extension into the temporal fossa.

All the teeth on the left side were vital. The author noticed, however, a marked recession on the gingiva over the mesial root of the first molar. Exploration with a probe revealed a communication with the maxillary sinus. Roentgenograms confirmed this finding. This established the etiology of the condition as a pyorrhetic sinusitis.

In retrospect Dr. Roy ventures the suggestion that the condition previously treated by him on the right side might have been due to the same cause. The temporal fossa complication, he believes, was probably due to an anatomical predisposition.

—*S. H. S.*

335—Schaer, Dr. Emile (Geneva): **Quelques cas cliniques de paradentose et leurs relations avec une modification du terrain.** Some clinical cases of paradentosis and their relation with a systemic disturbance, in: L'Odontologie, 75: 351-366. 1935.

The author is of the opinion that the state of the general constitution of the individual is the greatest factor in paradentosis. Local causes are secondary in nature. Nineteen cases are presented. In each case calcium and phosphorous determinations of the blood were made; as were complete x-ray and clinical investigations. Particular attention was paid to calcemia, phosphotemia, endocrine dysfunction, urinary analysis, acidosis and alkalosis. The author reports an analogy between para-

dentosis, osteitis fibrosis of Recklinghausen and Paget's disease.

Dr. Schaer's conclusions are as follows:

"Of the investigation of these cases we must keep in mind the importance of:

1. The metabolic dysfunction of Ca and P and their relation with the endocrine system. Also the hepatic dysfunctions.
2. The relative unimportance of local causes so often invoked: i.e., overhanging fillings, extractions, tartar, etc.
3. The great frequency of heterotropic calcifications in the pulp.
4. The symmetry of the alveolar lesions and the direction of their resorption in a mesio-distal direction.
5. The progressive extent of the bony lesions if the system is not treated.
6. The comparison of paradentosis with the first signs of Recklinghausen's osteitis fibrosa and Paget's disease. Out of 17 cases in which Ca-P determinations were made 2 were normal; 10 were lower and correspond to Paget's disease; 5 were higher and correspond to osteitis fibrosa. One case showed a hypocalcemia (78 mgs.) which was rapidly transformed to hypercalcemia (124 mgs.) by administration of parathormone. In general the blood P seems more variable than the calcemia."

—*G. H.*

336—Simonton, F. V., D. D. S.: **Periodontoclasia,** in: J. Periodontology, 6(1): 7-21. 1935.

A commendable and comprehensive survey of the manifold complexities in the etiology of periodontoclasia (dental periclasia), based on related literature and the author's own experimental investigation.

Dental periclasia is divided into two broad types; the inflammatory and the non-inflammatory. These are further subdivided according to the depth of penetration.

Among the factors associated with inflammatory periclasia involving only the gingiva are:

1) Stagnation of oral fluids. This is combated to some degree by function and to a minor extent by diet. Work done by Simonton in 1927 showed that even the so-called detergent diets were not effective in establishing mouth cleanliness:
   The toothbrush is effective.
2) Function: This was demonstrated by experiments on dogs.
3) Deficiency of food elements: Vitamins A. C. D. & G.
4) Mineral poisonings such as Hg, Pb, Bi, As, Cu.

An investigation into the mode of spread of Vincent's infection was made using a group of dogs as subjects. The author found that the disease was transmitted as an epidemic, but whether it attacks an individual has nothing to do with general or mouth health.

Returning to gingivitis, Dr. Simonton regards salivary calculus as an etiologic factor. This is of course associated with stagnation. Anatomical anomalies and faulty dental restorations are further factors.

An interesting development in the symptomatic treatment of gingivitis is the use of the Ouden current. This is a high voltage, cold current which can penetrate the tissues to some depth. It acts by stimulating the sympathetic nervous system which in turn causes an increased flow of blood to the part. The current can produce diathermic effects as well.

337—Sorrin, Sidney, D. D. S.: **Habit: An etiologic factor of periodontal disease,** in: D. Digest, 41: 290-298. 1935.

Dr. Sorrin calls to attention the importance of individual habit peculiarities of each patient as a factor in periodontal disease. These habit peculiarities are generally obscured by numerous local and systemic factors. It will be found oftentimes that when a periodontal condition will not respond to treatment, a habit peculiarity of the patient lies behind the disease. Dr. Sorrin for practical purposes groups the habits into three classes; those resulting from neuroses (lip biting, teeth clenching, tongue pressure); those arising out of one's occupational practice (holding nails in mouth, etc.,) and miscellaneous habits (pipe smoking, thumb sucking, etc.) Numerous photographs of pathological mouths resulting from habit peculiarities are presented.

—*G. H.*

338—Steiner, Otto: **Besprechung des kleinen Paradentosestatus und der Paradentosefragen im Lichte von Wissenschaft und Praxis.** Discussion of the narrow views of paradentosis and the questions of paradentosis in the lights of science and practice, in: Deutsche Zahnärtz. Wochensch., 38: 9-13. Jan., 1935.

The author discusses the etiology of paradentosis as presented today. These refer to local resistance, heredity, constitutional disturbances, nutrition, vitamins, Ca-P balance, constitutional and mental make-up as classified by the psychiatrists, nervous diseases.

He decries the ignorance and method of diagnosing paradentosis and calls for a more complete history and broader approach. He recommends taking of family history for hereditary clues, for constitutional questions, kind of previous paradentosis and results, former and present nervous disorders, hormone disturbances, and all possible general and organic diseases.

This he hopes will give a stimulating and more fruitful approach to the etiology of paradentosis.

—*N. K. B.*

339—Strean, L. P., D. D. S.: **Etiology of periodontal diseases,** in: D. Cosmos, 77(11): 1060-1067. Nov., 1935.

Effective treatment of a condition depends upon the detection of the cause.

The following is the author's summary:

"Before a cure of periodontal diseases is attempted, the cause must first be established. At the present time there does not seem to be one specific cause, but a combination of several predisposing and exciting factors. The causes may be local or systemic. Local causes are either mechanical, chemical, bacterial or a combination of these, the most prevalent being traumatogenic occlusion. The systemic causes may be attributed to improper diet, endocrine dysfunction, diabetes, pregnancy, various blood dyscrasias, etc. Laboratory tests, including blood count and blood chemistry, are very necessary in many cases before a diagnosis can be made."

—*S. H. S.*

340—Sutton, Harvey: **Notes sur la genise de la peri-odontite.** Notes on the origin of parodontoses (Med. Jour. of Aust. 11: 16. Oct., 1934). Abst., in: Revue Le Stomat.

Gingivitis is the first stage in parodontoses. It is most common in children and young adults. Its progress is slow and insidious. Out of 4,000 English children, 100 presented a marginal gingivitis up to 13 years of age. At 20, 100 had Vincent's spirochetes in the mouth.

Dr. Sutton claims that vitamin A keeps up the resistance of the tissues. A deficiency of vitamin A causing a hyperclasia and metaclasia of the mucous membrane.

During the formative period of the dental tissues, periodontal lesions can be initiated and arrested in dogs by controlling the amounts of vitamins A and D in the diet. Vitamins B and C are also important, especially vitamin B_2 in the resistance to Vincent's infection.

—*S. H. S.*

341—Wearn, E. E., D. D. S.: **Salivary and serumal calculus,** in: D. J. Australia, 7: 293-298. 1935.

Salivary calculus is the cause of simplex periodontitis; and it is impossible to clear up the pockets of complex periodontitis until all serumnal calculus is removed. There are two types of calculus, salivary and serumnal. The salivary calculus varies in hardness from soft whitish yellow on the buccal of the upper molars to a hard dark type on the lingual of the lower incisors. The hardness depends on the amount of its lime constituent to the mucin constituent. Serumnal calculus is found around the roots and is not of salivary origin but is the result of a primary inflammatory process. It is composed of dehydrated pus and lime salts from the blood.

Salivary calculus causes tissue atrophy and bone recession. No pockets are formed because of its presence. Serumnal calculus causes bone destruction because of its position, the soft tissues are not destroyed but deep pockets are formed.

The deposition of lime salts from the blood is induced by a reduction of CO_2, i. e. any condition causing anoxemia with more rapid breathing and a consequent washing out of CO_2. Removal of serumnal

calculus is by instrumentation and by correcting the salivary alkalinity. If the saliva is alkaline the blood acidity may be increased by such measures as exercise, diet, removal of worries, etc.

—*G. H.*

342—Weeks, H. W.: **Pedodontia to prevent periodontia,** in: Dental Outlook. Abst. from Review of Dentistry for Children, January, 1935: 22: 160. March, 1935.

Dentistry for children affords an opportunity to observe all lesions tending towards periodontal disease and a chance to prevent or check these tendencies. Beginnings of gingivitis are found in the child. Advancement of this condition leads to periodontal infection, alveolar destruction, and final tooth loosening. Proximal cavities and food impaction may cause gingivitis. The child then chews on the opposite side of the mouth to avoid pain to his sore gingivae, and forms a habit thereby which is the forerunner of periodontoclasia. Gingivitis on both sides may be found in children who are pasty-colored and poorly resistant to infection.

Other causes of gingivitis in children are.

1. Loosened deciduous teeth.
2. Malposed teeth.

Prophylaxis, restoration of function and gingival massage relieve this gingivitis in a great number of cases. The writer feels that there is as much periodontal disease in children as there is caries.

343—Weinman, Dr.: **Facteurs constitutionel dans l'étiologie et le traitement des maladies du paradentium.** Constitutional factors in the etiology and treatment of paradental diseases. Abst., in: Rev. de Stomat, 37(10): 694-695. 1935.

The theory is advanced that pyorrhea starts when the cementum loses the power of stimulating the periodontal connective tissue to lay down new cementum. The loss of this power and consequently the initiation of the disease is due to a disturbance in metabolism, involving a decrease in the specific dynamic action of proteins and in an increase of blood, cholesterin and sugar in the blood.

The patients present symptoms of amenorrhea, adiposity, hypertrichosis, dry skin, impotence, etc. These symptoms may be related to dysfunction of the anterior lobe of the pituitary gland.

X-ray radiation of the pituitary gland can be employed in treatment.

Favorable results were also obtained by insoufflation of oxygen.

—S. *H.* S.

344—Weiss, L. R.: **Development and pathology of periodontoclasia,** in: Minn. Dist. D. J., 18:20. Mar., 1935.

This article presents a condensed review of pyorrhea. The author goes into the histological and clinical pictures, the etiology and pathological symptoms and types of pyorrhea.

Both the curettage method of Riggs and the histological studies of Gottlieb, Kronfeld, Orban and Box, are mentioned as fundamental to a complete understanding of the pathosis of pyorrhea.

The author retains the following definition of pyorrhea: "A suppurating inflammation and absorption of the tissues surrounding the teeth." The symptom picture in pyorrhea from a chronological standpoint is: gingivitis, periodontitis, absorption of the alveolar process, gingival recession, pocket formation, suppuration, and tooth mobility. Not all of these must take place to diagnose the condition as periodontoclasia.

Types:

1. Horizontal
2. Vertical

Causes:

1. Systemic
   a. Avitaminosis
   b. Diabetes
   c. Progressive age disturbances
   d. Chemical poisoning
2. Local conditions
   a. Loss of function
   b. Food impaction
3. Lack of mouth hygiene
4. Occlusal imbalance

Pathology:

1. Marked hyperemia
2. Thickened peridental membrane
3. Osteolysis

Pathological Types:

1. Prurulient
2. Low grade aprurulient granulomatosis

The author feels that a complete knowledge of the foregoing phases of pyorrhea must be had to obtain clinical success.

(e) Symptomatology and Diagnosis

(Continued from 6 (2): 90. July, 1935)

233—Ackermann, Francois: **Normal dental articulation (occlusion).** Its local, regional, and general modifications, in: Rev. de Stomatol., 37: 353. June, 1935. Abst., in: J. Am. Dent. A., p. 1625. 1935.

Believing that there have been no general articles on the normal occlusion, the author undertakes to present and summarize all the known facts, beginning with a presentation of the nomenclature which is based on biophysiomorphology. He points out four periods or conditions of occlusion: 1. Normal occlusion may persist throughout life in a healthy condition. This assumes that the pressures and contrapressures have maintained a state of equilibrium between the teeth, parodontium and the general state. 2. The normal occlusion usually undergoes a period of biologic unstable equilibrium. In this period, one may note a marginal gingivitis accompanied by diffuse alveolar atrophy. The etiology may be humoral, endocrine, toxic, vagosympathetic, mechanical, etc. 3. The normal occlusion may undergo a period of real biologic deficiency. Insufficiency of the supporting tissues is manifested by dislocation of the occlusion. 4. The normal occlusion may undergo a period of early decline, which the author calls precocious senescence. Here, there may be two aspects, one of extreme erosion of the teeth without parodontosis; the other, of relative erosion of the teeth and atrophy of the supporting tissue. Changes in normal occlusion afford biophysiomechanical information of great interest and significance to the practitioner. —*T.*

234—Allen, J. B., D. D. S.: **Value of early diagnosis and treatment of ulcerative stomatitis in children,** in: Intnat'l Jour. Orth. & Dent. for Children, 21(8): 749-755. 1935.

To help maintain the general health of the child, it is essential that the dentist makes a thorough examination and early diagnosis of all ulcerative conditions in the oral cavity.

Dr. Allen summarizes the symptomatology and treatment of the different forms of stomatitis, including (a) simple stomatitis (b) traumatic stomatitis (c) parasitic stomatitis (thrush) (d) Herpetic stomatitis (e) Perleche (a contagious disease of bacterial origin characterized by deep cracks or fissures at the corners of the mouth. Treatment consists in applying 7% tincture of iodine and aconite to the affected part. (f) Apthae (g) Stomatitis due to difficult dentition.

The dentist should be quick to recognize the oral manifestations of contagious diseases. "As a general rule, ulcerations in the oral cavity precede most of these manifestations." The oral symptomatology of the following conditions is presented: (a) Vincent's stomatitis (b) syphilis, primary, secondary and tertiary (c) Scarlet fever (d) Measles (e) Diphtheria (f) Metallic poisoning—Lead, Mercury, Phosphorous, Bismuth, Copper. (g) Medicamentous stomatitis (h) Allergic stomatitis.

—S. *H.* S.

235—Becks, H., M. D., D. D. S., and Fleming, W. C., D. D. S.: **Is osteodystrophic type of paradentosis, as produced in animals, identical with one form of paradentosis in man?** Abst., in: J. D. R., 15: 206-207. 1935.

The authors supplement the results of Becks and Weber with clinical findings in the oral cavity of dogs that have received diets low in Ca. and vitamin D. Roentgenograms were used as an adjunct in diagnosis. The type of paradentosis noted is designated as osteodystrophic and is compared with a similar lesion in man characterized by the following conditions:

1) Changes in the alveolar structure and "bony part of the paradentium," which are osteoporotic. This is manifested in the roentgenogram by a peculiar radioluscent appearance of the alveolar bone.

2) Lack of inflammation at the beginning of the disease.

3) Teeth become loose and tend to migrate.

4) Usually find increased Ca., normal or low inorganic P. of blood serum; also thyroid dysfunction and dietary deficiencies.

5) Generalized root-end resorption occurring frequently.

The above type is not similar to Gottlieb's diffuse atrophy. It constitutes an outstanding type of paradentosis in man.

—S. *H.* S.

236—Beregoff, P., M. D.: **Infection of the mouth in relation to dysentery with report of a case of intestinal amebiasis,** in: Canadian Med. Ass'n Jour., 32: 639-641. 1935.

The author brings up the question of whether amebiasis of the mouth is a predisposing factor of intestinal amebiasis. He suggests the possibility, if such is the case, of considering amoeba gingivalis a preliminary form of entamoeba histolytica. In a previous study cats fed with cultures of amoeba gingivalis presented an attenuated form of dysentery. Furthermore, in studying pyorrhea cases in South America it was found that all cases studied presented amoebae in the gingival pockets and in 50% of the cases the amoebae closely resembled the entamoeba histolytica.

A case report of a young woman having suffered for years with sore gums and loose teeth is presented. The author concludes that "the careful history of the patient's environment, the hygienic condition of the home, the lack of the same infection in any other member of the family, and the history of a long standing mouth infection, suggest that the predisposing factor of the intestinal condition was the mouth condition.

—*G. H.*

237—Cannon, A. B., New York: **White sponge nevus of mucosa (naevus spongiosus albus mucosae).** Archives of dermatology and syphilology, Chicago, Mar., 1935. Abst. (condensed) J. A. M. A., 104(17): 1556. Aug. 17, 1935.

Cannon found peculiar whitish lesion in mouth of woman examined because of a banal skin eruption. Further investigation revealed similar discoloration of the labia, the vaginal walls and upper part of anal canal and rectum. Several members of family had similar mouth condition. Cancer was feared because of family history of many fatal malignancies but present condition is not to be considered even potentially cancerous unless some unexpected change occurs in affected area in some member of the family. Contrary to the part played by smoking in leukoplakia and in certain malignant types of degeneration of the tongue, in this case no slightest change followed smoking of as many as twenty cigarettes a day over a period of two years. Absence of subjective symptoms and of any signs of inflammation, static nature of process and its limitation to above regions, along with its congenital hereditary familial basis suggest its classification among the nevi. In the absence of a recorded parallel case author proposes name "White Sponge Nevus of Mucosa."

238—Davis, Wm.: **Bleeding gums,** in: N. J. State Dental J., 7: Special issue: 39-41. 1936.

The article is a copy of a radio talk designed to acquaint the layman with the significance of bleeding gums in pyorrhea, improper tooth contour and Vincent's disease. The importance of early and regular dental care is stressed.

—*G. H.*

239—Delagnes, George, D. D. S.: **Paradentosis, classification and diagnosis,** in: Dental Survey, 11: 32-34. Jan., 1935.

In 1931, the International Association for Research in Paradentosis was organized. The Swiss section of this association presented a compact classification for which refer back to last year's periodicals.

240—Epstein, I. A., D. D. S.: **Clinical indications for the use of blood examinations in the practice of periodontia,** in: J. Periodontology, 6: 30-41. Jan., 1935.

An excellent survey of the blood dyscrasias showing symptoms in the oral cavity. It is imperative that the dentist recognize these conditions, inasmuch as he is often the first one to see the patient in the early stages.

The various types of blood diseases, are presented with their oral clinical symptoms, as well as the means of making a differential diagnosis. The blood pictures are characteristic for each disease.

Among the conditions described are: anaemia pernicious and secondary; leukaemia, lymphatic, myelogenous, acute; Vincent's infection; scurvy; purpura; agranulocytosis; haemophilia; aplastic anaemia; metallic poisonings; Hodgkin's disease.

The article is prefaced by a brief account of the general features of the origin, relation and functions of blood cells.

The author closes with the statement that "the periodontist has the right and is distinctly obligated to order examination of the blood in cases in which he believes blood findings to be of value, just as he makes use of other diagnostic aids at his command."

—S. *H.* S.

241—Figi, Frederick A.: Diseases of the soft tissues of the mouth, in: J. Am. Dent. A., 22(11): 1919. Nov., 1935.

There are many local and systemic conditions which may be detected in their incipiency by the dentist. A variety of lesions, inflammatory, metabolic and neoplastic in nature occur in the soft tissues in the mouth. These vary in progress, occurrence and prognosis. The diagnosis of such lesions is often more important to the dentist than their treatment. Gingivitis, including Vincent's infection, through differential diagnosis, which must include laboratory findings, ought to be distinguished from symptoms of blood dyscrasias. Agranulocytosis, scurvy, and sprue must be distinguished from Vincent's infection.

Pathologic changes occur in the mouth from granulomas, syphilis, tuberculosis, actinomycosis, blastomycosis, rhinoscleroma and leprosy. The author gives descriptions of lesions typical of these conditions as they appear in the mouth.

Thrush of the acute or chronic type may occur in mouths of children under five years or of old people. Diagnosis is made from films of the membrane and cultures of the organism.

Stomatitis in the mouth may also be of aphthous or herpetic types. Etiologies are from primary or secondary anemias, chemicals, or drugs such as lead, bismuth, mercury, and phosphorous.

Geographic tongue and Fordyce's disease are conditions to be recognized and left alone.

Leukoplakias of three types are described. 1—Squamous (benign). 2—Papillary (potential neoplasm). 3—Syphilitic (often neoplasmic).

Lingua nigra (hairy tongue) is described.

Pemphigus, a bleb-forming disease, may be easily diagnosed from oral symptoms. It should be distinguished from erythema multiforme.

Neoplasms of benign and malignant nature are mentioned. These include epulides, hemangiomas, lymphangiomas, fibroma, adenoma, papiloma, and mucous and dermoid cysts. Malignant types, epithelioma and sarcoma are also mentioned.

This article presents a very practical approach to oral lesions because of its orderly arrangement and helpful hints towards adequate diagnosis.

242—McGeorge, J. R., D. D. S.: Mercurial stomatitis, in: J. Am. Dent. A., 22: 60-64. 1935.

A brief presentation of the etiology, pathology, symptoms, diagnosis, prognosis, prophylaxis and treatment.

The following is part of the author's summary:

1) Mercurial stomatitis may be induced industrially and therapeutically. It usually results from the use of medicines containing some form of mercury.
2) Some persons are very susceptible to the toxic action of mercury.
3) The pathological process has not been definitely determined. It may be direct corrosion by mercury in the saliva or possibly a super-induced infection in a field of reduced resistance.
4) Stomatitis becomes very severe if allowed to progress. Sloughing and ulceration occur on the tongue, lips, gums and cheeks. Teeth loosen and are covered with sordes. The tongue is swollen and there is a continued drooling of saliva.
5) Extraction is contraindicated during the acute stage.
6) Clinical picture resembles Vincent's infection. The odor is characteristic, the blue line is not.
7) Poor mouth hygiene is a predisposing factor in mercurial stomatitis, which never occurs in an edentulous mouth.
8) Local treatment should be palliative. The administration of Hg should be stopped, the system alkalinized and diet regulated.

—S. *H.* S.

243—Ginander, G. J., M. D.: Eruptive fever with stomatitis and ophthalmia, in: Am. Jour. Dis. of Children, 49: 1148-1160. 1935.

Periodically there have been reports in the literature of sporadic cases of exanthemata in children, with involvement of the buccal and ocular mucosa. These cases present syndromes which conform with no known clinical entity. They have, for want of fuller knowledge, been referred to as atypical manifestations of erythema exudativum multiforme (Hebra). The generalized clinical findings have been summarized by Stevens as follows, "Here is a syndrome of dramatic onset, with fever, conjunctivitis and cutaneous eruption. The child is prostrated, the mouth and tongue are raw and inflamed, the eyelids are swollen and pus streams from the eyes . . . . ."

The author presents a case falling into this same group. In addition to the generalized skin eruption and ophthalmia the mouth lesions are of interest. The entire buccal mucosa, including the tongue, hard and soft palates, were red, ulcerated in spots and covered with a whitish pseudo-membrane, which was friable and appeared like ruptured bullae. The lesions varied in size and were surrounded by a zone of marked congestion. The gums were swollen and inflamed and showed the presence of old and fresh blood at the margins.

With frequent sodium perborate washings the mouth showed an early improvement.

—*S. H. S.*

244—Hulin, Ch.: **Presentation d'une fiche d'observation et de traitement des paradentoses.** Presentation of a chart for the observation and treatment of paradentoses, in: L'Odontologie, 73: 10-17. Jan., 1935.

Dr. Hulin presents a record chart which has the double purpose of aiding in the determination of the general disturbance of which the paradentosis is a manifestation; as well as a detailed record of the oral lesions and their treatment. Under the clinical description Dr. Hulin employs the scheme Fournet evolved for recording the exact location of pockets, the amount of pus and the degree of mobility. Each root is portrayed as concentric circles divided into four parts according to mesial, distal, lingual, and buccal. Mobility and pus are represented by M and P and are recorded from 1 to 5, according to degree. The anatomical gingival contour is also noted.

Since paradentosis is a symptom of a systemic disorder the chart has an important place for the recording of arthritic symptoms, hepatic dysfunction, and the patient's diet. Urine and blood analysis are also recorded. Treatment is recorded last.

—*G. H.*

245—Knapp, E.: **An unusual case of periodontitis marginalis progressiva chronica in a thirteen year old girl;** in: Deutsche Zahnärtz. wochenschr., 38: 1080-1081. Nov., 1935.

The thirteen year old girl was presented to the dentist complaining of a strong periodontal pain in her lower right first molar. On examination all the other first molars were found to be carious and had to be extracted. This was deferred until the second molars erupted.

On examination of the whole mouth an extensive hypertrophic, reddish condition of the interdental papillae in the upper incisor region was noted. There was distinct mobility and pocket formation, plus pus. X-ray showed bone rarefaction, two-thirds of the length down the root.

A year before she had suffered a fall which produced loosening of the incisors. The author was convinced that the fall alone could not have produced such an extensive lesion, especially at this age. Some endogenous forces probably played a part. In reviewing all possibilities he came to the conclusion that entering the state of puberty and the chlorosis present brought about a disturbance in the body which was precipitated by the fall.

—*N. K. B.*

246—Leabo, W.: **Problem of periodontal disease,** in: Impressions, Summer, 1935. Abst., in: Dental Outlook, 22: 396-397. 1935.

The writer states that diagnosis is of infinitely more importance to the general practitioner than to the specialist, for the simple reason that the patient coming to the specialist has already been informed of a certain amount of trouble, while the number of people who are seeing their dentist periodically with periodontal lesions of more or less extent, are numerous. The latter have no idea of the dangers present, due to the failure of the dentist to recognize the early clinical appearance of the disease

present. It is remarkable how many cases of dental periclasia are discovered when radiographs are taken, neither the patient nor the dentist being aware of the disturbance. It is not conceivable how prophylaxis can be given without discovering the breaks in the gingival attachment characteristic of periodontal lesions, and the detection of subgingival deposits. It is not uncommon for a patient to develop an acute periodontal abscess before he is aware of any trouble, even though he is a regular visitor to his dentist for fillings and having his teeth cleaned. From a consideration of these important points in diagnosis, the operation of prophylaxis takes on increased importance, and carries with it a responsibility which we can not take lightly.

247—Leonard, H. J., D. D. S.: **Diagnosis of conditions leading to destruction of the teeth and surrounding structures,** in: D. Cosmos, 77: 276-281. 1935.

Years of intensive research on dental caries indicates that caries susceptibility is a local manifestation of some metabolic irregularity. Optimal diets rich in Ca, P, and vitamin D, may temporarily reduce caries susceptibility but it has not been proven that caries susceptible individuals are deficient in Ca or P, by any chemical tests. Treatment along systemic lines at the present time is negative. The solution to treatment of caries lies in its prevention by early operative treatment and proper toothbrush care.

Diseases of the soft tissues of the mouth are rarely, purely systemic in origin and even when systemic factors do exist, diagnosis and treatment of the local etiologic factors will suffice to bring about a cure. Metabolic deficencies, endocrine dysfunction, and toxic materials in the blood (Hg, As, uric acid in nephritis, sugar in diabetes, etc.) predispose to periodontoclasia. Overeating also acts as a predisposing factor to periodontoclasia by acting thru the saliva in the deposition of irritating toxic material on tooth surfaces. These systemic factors act by aggravating the local conditions. The three great local causes are infection, trauma, and lack of function. Early recognition of calculus, food impaction areas, restoration of missing teeth, and proper home care are the most effective means of preventing periodontal disease.

—*G. H.*

248—Mackie, Thoma T., M. D., assisted by Miss Madeline Henriques, New York: **Ulcerative colitis. II—The factor of deficiency states,** in: J. A. M. A., 104 (3): 176. Jan. 19, 1935.

The authors' studies of 75 consecutive cases of chronic ulcerative colitis led them to believe that deficiency states play an important role in mechanism of the disease; 62.6% of cases studied giving evidence of deficiency states. The diagnostic criteria adopted were restricted to the objective changes definitely known to be associated with various deficiency states. Among the most prominent of these and one of the earliest recognizable is the alteration in mucous membrane of the tongue. Starting with inflammation of the anterior fungiform papillae, a diffuse glossitis develops if condition is not checked. Small painful aphthous ulcers develop on tongue and may be present also on buccal mucosa. Ultimately smooth atrophy of the tongue results. Very rarely in advanced serious cases mucous membrane of mouth becomes involved to such an extent as to be indistinguishable from the diffuse stomatitis pellagra. In a series of 47 cases, authors found tongue symptoms in 46, and involvements of buccal mucosa in 4. In a tabulation of oral lesions associated with the disease under study, in a series of 46 cases, they found inflamed lingual papillae in 31; aphthous ulceration, 8; smooth atrophy of tongue, 29; diffuse stomatitis, 4. Inadequate supply of the vitamin B complex seems to be a significant factor in these mouth lesions. Liver preparations administered orally or parenterally have cured tongue condition; and the same preparations given intramuscularly have checked aphthous ulcers and diffuse stomatitis.

249—Martin, Eric: **Les variations de la calcémie dans les états pathologiques et leur signification.** Calcium variations in pathologic states and their significance, in: L'Odontologie, 75: 769-779. 1935.

The determination of the blood Ca is indispensable in treating bone diseases. It represents, however, only one of the diagnostic factors. Normally it does not allow

of any conclusion. Pathologically it is characteristic of no osseous disease.

—*G. H.*

250—McCarthy, F. P. and Dexter, S. O. Jr.: **Oral manifestations of bismuth,** in: New Eng. Jour. of Medicine, Aug. 22, 1935. Abs. (condensed) from J. A. M. A., 105(17): 1384. Oct. 26, 1935.

McCarthy & Dexter noted an incidence of eighty per cent bismuth pigmentation of mouth in a series of five hundred and eleven hospital cases treated for syphilis. Indications all point to the fact that oral hygiene and tartar deposits represented the important factor in this high percentage. A series of urinary examinations in severe pigmentation cases was negative for nephritis, and blood examinations showed no dyscrasia related to the bismuth therapy. In contrast, a small control group in private practise showed only about five percent pigmentation. Removal of tartar deposits with improved dental hygiene hastens disappearance of pigmentation and tends to prevent future discoloration. Since using (for ten years) insoluble bismuth salicylate in oil, necrosis and gangrene have been rare and severe gingival pigmentation and inflammatory reactions relatively few.

251—Merritt, A. H., D. D. S.: **Vincent's infection and periodontoclasia.** The secret of successful treatment, in: Jour. N. Y. Acad. of Dentistry, II (2): 41-50. 1935.

Clinically there are two types of Vincent's infection, acute and subacute. The latter resembles chronic gingivitis, superficially, but is differentiated by being associated with pain. The predisposing factors are considered to be oral sepsis and excessive smoking. Treatment consists in the establishment of hygiene and the use of an oxidizing solution.

Periodontoclasia is manifested by the destruction of the alveolar crests of bone with the subsequent formation of pockets. Systemic conditions play a minor role in the etiology, and this through a disturbance of the acid-base balance of the blood and tissues. Local conditions causing a predisposition to periodontoclasia are legion. They are: (a) malocclusion, loss of teeth, malfunction, etc.

Treatment falls under four main divisions, (a) local operative procedure, (b) balancing occlusion, (c) systemic treatment, (d) post operative care. Under operative procedure are mentioned subgingival curettage, flap operation, modified flap operation and gum resection. Systemic treatment includes adjustment of diet, blood examination and coöperation with physician.

—*S. H. S.*

252—Moreau, R., Debray, Chas.: **Sur un cas de gingivite scorbutique.** Concerning a case of scorbutic gingivitis, in: L'Odontologie, 75(12): 829-832. 1935.

The authors report an interesting case of a patient, male, aged 50. The man was confined to bed suffering from an affection characterized by a hemorrhagic syndrome. There were large purpuric hemorrhages, especially in the lower part of the legs. There was a general debility, asthenia and anemia. The gingivae were inflamed, partially gangrenous, bleeding readily. The teeth were loose.

Clinical tests corroborated the diagnosis of scurvy. The diet was found to be deficient in vitamin C.

An adequate diet, containing abundant vitamins was prescribed and the patient made a speedy recovery.

Three weeks later the patient presented again. This time he had none of the classical symptoms, only a hypertrophic gingivitis. The teeth were loose but there were no other symptoms. Clinical tests again demonstrated scurvy. Upon questioning it was learned that he had recently gone back to his old vitamin C. deficient diet.

The point brought out is that had it not been for the previous clear cut scorbutic condition the second mild attack without classical symptoms would in all probability never have been correctly diagnosed.

The authors contend that many cases of sub-clinical and early scurvy remain unrecognized.

—*S. H. S.*

253—Nathanson, Israel: **Infectious diseases of the mouth,** in: D. Cosmos, 77: 502. May, 1935.

The author lists and describes the oral findings present in infectious diseases. The

mouth being an ideal culture haven, provides medium for many micro-organisms which may produce local and systemic disturbances. Familiarity of the dentist with such lesions and the supplementary aid of a physician may prevent serious manifestations, if recognized and treated early. Once noted, the differential diagnosis then becomes important for accurate recognition.

1) Vincent's infection:
   a) symptoms:
      1) bleeding gums
      2) grey-white pseudomembrane
      3) interproximal papillae enlarged
      4) increased salivation, temperature rise, malaise
      5) loosened teeth
   b) causes: are predisposing local conditions, i.e. poor dentistry, poor hygiene, tobacco, alcohol, and parodontosis.
   c) differential diagnosis:
      1) confirmed by microscope
      2) distinguish f r o m poisonings, blood dyscrasias, pemphigus, diphtheria, gingivitis, syphilis, tuberculosis, etc.
   d) treatment:
      1) local—oxidizing agents, like hydrogen peroxide
      2) general — injection of sulpharsphenamine and cyanide of mercury.
2) Gonorrheal Stomatitis:
   a) symptoms:
      1) mucosa and gingivae inflamed, painful bleeding; purulent discharge about the teeth
      2) ulcers on the tongue and lips
   b) diagnosis; is microscopical, showing a gram-negative intracellular diplococcus
   c) differential diagnosis:
      1) history of drug administration
      2) Vincent's infection by microscopical examination
3) Diphtheria:
   a) symptoms:
      1) typical pseudomembrane formation
   b) diagnosis: Loeffler medium growth
   c) differential diagnosis: distinguished from Vincent's, Thrush, scarlet-fever, diphtheria, and follicular tonsilitis
4) Foot and mouth disease: vesicles on the mucosa which heal without scarring. History of occupation.
5) Thrush: milkwhite patches on the palate, gums, and buccal mucosa
   diagnosis: microscopic picture shows Oidium albicans
   differential diagnosis: diphtheria and aphthae
6) Tuberculosis: small yellow nodules which break down, ulcerate, leaving a dark red border.
   diagnosis: secondary to respiratory tuberculosis
   differential d i a g n o s i s: distinguished from syphilis by a Wasserman test
7) Actino mycosis: tumor-like swellings
   diagnosis: sulphur granules
   differential d i a g n o s i s: distinguished from sinuses with suppurative condition
8) Glanders: abcesses with sticky fluid
9) Syphilis: Stage 1, 2 and 3
   diagnosis: Wasserman test
   differential diagnosis: yaws and gangosa
10) Leprosy: nodules on the mucous membrane
11) Sprue: vesiculation and ulceration of the tongue. Fungiform papillae swollen
12) Yaws: multiple granulomatous lesions and whitish plaques
    Tropical disease
    Distinguished from syphilis
13) Gangosa: Ulceration of perforation of the palate.

—*S. E.*

251—Robinson, H., L. D. S., R. C. S.: **Pyorrhea, an attempt to differentiate clinical types with suggestions for nomenclature,** in: Dent. Mag. and Oral Topics 52(9): 855-857. 1935.

The author differentiates clinical types of parodontoses and suggests descriptive terms to prefix pyorrhea. There are four types, as follows:

Type 1—Ortho pyorrhea—this embraces pyorrhea alveolaris in its classic form.

Type 2—Hypertrophic pyorrhea—the gums are pale, papillae and marginal gingivae hyperaemic and hypertrophied. There is no alveolar crest resorption. This type is seen in younger persons.

Type 3—Rubro-pyorrhea—seen in association with well formed teeth showing marked attrition. There is a uniform redness of the gums which are firm and appear to be stretched.

Type 4—Senile pyorrhea—This is the pyorrhea of old age. There is a physiological alveolar absorption complicated by peridental inflammation. There is little pus and the pockets are shallow.

—*S. H. S.*

252—Schlutz, Frederic W., M. D.: **Systemic thrush in children.** J. A. M. A., 105(9): 650. Aug. 31, 1935.

Thrush is a fungoid disease belonging to a group of mycoces which affect not only the skin but also the mucous membranes and internal organs and in rare cases cause general sepsis. Intestinal secretions do not destroy the organisms and although thrush probably *always starts in the mouth,* the entire alimentary tract may become involved. Thrush is not contagious. It is usually confined to infants, but may occur at any age. The common form of oral thrush is found principally in poorly nourished children and this mold disease usually responds readily to simple treatment. Sometimes, however formidable symptoms develop with clinical aspects of general infection extremely resistant to therapy, and then fatal termination is likely. It is not known why this usually localized, superficial disease, after remaining latent, sometimes flares up into a very serious infection. Dr. Isaac Abt (discussing) says that it is to be assumed that infection occurs only when there has been *injury* to *oral mucosa* or when its resistance has been lowered. General resistance is probably very low when thrush infection invades blood stream and Dr. Abt surmises that the importance of the role of this infection in some of the severe and fatal diarrheal diseases may have been underestimated. Schlutz reports four cases of systemic thrush out of a total of only five cases which he has seen during twenty-five years of pediatric practice. All of these showed very unusual symptoms. In two there was pronounced dwarfing—and in three, a general alopecia with complete and permanent loss of all the hairs of face and scalp. In one, an infant of four months, the sepsis developed with amazing rapidity and no local or systemic therapy availed to check progress of disease to fatal culmination.

(*Italics not in original.*)

253—Simpson, C. O., M. D., D. D. S.: **Radiographs in periodontia,** in: Dental Radiography and Photography, 8:1. 1935.

The author believes that radiography is the dentist's chief ally in recognizing and maintaining a healthy supporting structure of the teeth. After listing possible causes of periodontoclasia:

1) malposition of teeth
2) occlusal trauma
3) nutritional disturbances
4) degenerative diseases
5) masticatory habits
6) senility

he discusses the value of radiography as a diagnostic device. Radiography, in a severe involvement of periodontal tissues, predetermines the height of gingival excision, and location of the necrotic areas requiring curettage. Since a loss of one-half the normal bone height or involvement of the bifurcation suggest a poor prognosis, the radiograph is indispensable to such diagnosis. Radiographs reveals the depth of paradontal pockets for any instrumentation which may follow.

In order to be able to make diagnoses of these conditions certain precautions must be taken in the radiographic procedure:

1) radiographs should be made annually to discover gradational bone changes
2) radiographs should be taken parallel to the approximal surfaces
3) a fifteen degree distal angulation is necessary for upper molars to disclose interradial resorption
4) bite-wing films reveal calculus but exaggerate the depth of advancing periodontoclasia
5) bite-wing films show the alveolar crest low (two mm. from the enamel margin)
   Periapical films show the alveolar crest at the height of the enamel margin
6) the height of the septal bone is in inverse proportion to the width of the interproximal space.

He concludes with a statement that

radiographs reveal tissue changes years before clinical manifestations appear.

—S. E.

254—Skosovsky, Dr. J., Strarbourg: **Recherches stomatologiques sur le pH intrabuccal.** Stomatological researches on the intraoral pH, in: L'Odontologie, 75: 712-713. 1935.

Using an electrometric technique copied after Vles, the authors measured the salivary pH in situ, i.e. intraorally. In the great majority of individuals the pH was found to be slightly acid. Three acidic zones were observed: the opening of Stenson's duct, that of Wharton's duct and the upper vestibule. The lower vestibule was found to be an alkaline zone. The values vary from day to day in the individual.

Pyorrhetics presented an acidity more marked than in normal subjects, while the carriers of gingivitis showed in the beginning an alkalinity. Caries was frequent where acidity was marked. The authors present no experimental evidence. The article is merely a statement of the above facts.

—G. H.

255—Tishler, B., D. M. D.: **A case of seventh nerve palsy associated with gingival and throat inflammation and fuso-spirochetal infection,** in: J. of Periodontology, 6: 43-46. 1935.

The author presents a report of a case of 7th nerve palsy associated with a fuso-spirochetal infection. The mucous membranes of the mouth were normal except for a whitish area on the lower right buccal mucosa and the right tonsillar region. There were no lesions typical of Vincent's infection but a smear revealed a multitude of the fuso-spirochetal organisms. The patient was in a debilitated condition, ran a temperature of about 99.5° plus, suffered from paralyzing of the facial nerve on the right side, cough.

Systemic treatment consisted of a nutritional diet (milk, orange juice, etc.), and rest. Local: The mucous membranes were swabbed with acri violet (2%); also sodium perborate was used as mouth wash. The oral condition cleared up in a week's time. (Previous to author's care, the patient had been under a physician's care for three weeks without showing any signs of improvement.) The palsy cleared up in 3 weeks.

—S. H. S.

(f) Treatment

(Continued from 6 (2): 100. July, 1935)

369—Aiguier, J. E.: **Dental care of the diabetic patient,** in: J. N. Y. Academy of Dentistry, 2: 99-105. Abst., in: D. Outlook, 22: 517. 1935.

Summary:

1. The physician should place the diabetic patient under the care of a dentist as soon as possible, in order that the mouth conditions may be treated.
2. The patient's teeth must be adequate for proper mastication of food, or artificial substitutes should be provided.
3. Diabetic pyorrhea and other infections of the mouth in diabetes can not be successfully controlled without treatment of the diabetes.
4. Loose teeth should not be removed if it appears possible to save them. Whenever possible extractions should be postponed. Frequently teeth will tighten after the diabetes has been controlled.
5. Diabetic patients should have a complete roentgenographic study of their teeth at least once a year, since the supporting structures of the teeth in the diabetic are more prone to become involved because of lowered resistance.
6. In caring for the diabetic patient, the tissues must be handled gently. The tissues have poor resistance in patients with high blood sugar, especially if associated with arteriosclerosis, as often occurs in the diabetic over fifty years of age. The tissues of the feet and toes have particularly low resistance, those of the oral cavity a little more.
7. If extensive dental work must be done it is better to do it in several stages.
8. Diabetics should have their mouths examined more frequently than others—at least every three months. Diabetic children require examination even more frequently—every two months.

370—Becks, Hermann, M. D., D. D. S., and Simmonds, Nina, Sc.D.: Dental caries and paradental disturbances. Importance of adequate diet for health of teeth and paradentium. Abst., in: J. D. Research, 15: 207. 1935.

Perhaps the lack of success in preventing caries and paradentoses in children by the administration of diets is due to the failure to examine the children for other possible (endogenous and exogenous) etiologic factors.

The authors found that a survey of 200 dietary histories "revealed high percentages of deficiencies in fundamental nutritional elements." This led them to work out 4 different types of diets, which would provide all the necessary food elements for the proper formation of the teeth and bones. "If nutrition *is* important for the health of teeth and paradentium, these diets, when conscientiously followed by patients, will be adequate—and will be caries preventive where only nutritional factors are at fault."

—*S. H. S.*

371—Belding, P. H., D. D. S., and Belding, L. J., M. D.: Physiologic dentistry, in: D. Cosmos, 77: 172-178. 1935.

The authors approach to periodontal disease is one of maintenance of normal gingival tone and its restoration if morbid; rather than a mere treatment of symptoms. They consider gingivitis as a precursor of pyorrhea, a specific disease due to the fuso-spirochetal symbiosis. Gingivitis, Vincent's infection, and pyorrhea are all caused by this bacterial complex; but represent different degrees of activity in the progress of the fuso-spirochetal infection.

Seventy-five percent of all adults have gingivitis and yet it is usually overlooked; and even when recognized its significance is not fully comprehended. Every case of gingivitis is a forerunner of Vincent's infection of the gingivae and eventually pyorrhea. The primary therapeutic effort should, therefore, be in the direction of eradication of the fuso-spirochetal infection by enhancement of resistance, both local and general. This includes restoration of oral hygiene, attention to proper diet, proper supervision of toothbrush technique, daily use of a spirocheticidal dentifrice or mouthwash, and general hygienic welfare.

The authors are of the opinion that the dentist, if he deals only with his own particular field in dietary problems, is capable of assisting in the direction of a normal balanced diet and may advise the prescription of such a diet.

The prevention of gingivitis is the prevention of periodontoclasia and consists of maintenance of the parts in normal physiologic health by balanced diet and general hygiene.

—*G. H.*

372—Belding, P. H., D. D. S., and Belding, L. J., M. D.: Specific treatment of periodontal disease, in: D. Digest, 41: 216-220. 1935.

The authors consider that "gingivitis, acute and chronic, pyorrhea and Vincent's infection are merely manifestations of the same disease, and that all are specifically caused by the activity of the fuso-spirochetal complex and cured by spirocheticidal therapy." As in all bacterial infections, exposure, fatigue, malnutrition, or any other factor that depresses resistance, either local or general, predisposes to the disease. The therapeutic treatment is, therefore, antispirocheticidal plus the removal of local and general predisposing factors. A 12.5% solution of bismuthous oxide organically combined, neoarsphenamine, or bismuth ion incorporated in a dentifrice may be used. Tables illustrating the nature of the gingivae, bone resorption and the treatment of the chronic periodontal diseases are presented. The chronic gingivitides are divided into three classes: 1) periodontal disease without pockets; 2) pockets with horizontal resorption of bone; 3) pockets with vertical bone destruction.

—*G. H.*

373—Bödecker, C. F., and Lefkowitz, W.: Gingival re-attachment to the enamel and its relation to operative dentistry, in: Trans. N. Y. State D. Soc., 115-123. 1935.

The discovery by Gottlieb of the normal attachment of the gingival epithelium to the enamel suggests that harm may result from operative procedures which sever the epithelial attachment. Bödecker and his coworkers have corroborated Gottlieb's discovery. They maintain that clinical demonstration of the attachment in man is

difficult because an instrument can readily be passed through this delicate epithelial attachment to the cemento-enamel border thus giving the impression of a gingival crevice. Histological evidence indicates the absence of any noticeable gingival crevice.

The transplantation of six teeth in five dogs with consequent normal reattachment of the gingiva to the enamel was accomplished. The likelihood of a similar reattachment of non-pathological tissue in man is indicated by the similarity of dog's gingiva to that of man. The clinical "tightening up" of the gingiva around fillings is an observed fact. Tissues loosened by operative procedures will become reattached to the cervical enamel.

This suggests the possibility of gingival re-attachment after paradentosis. Whether there is an actual re-attachment or a shrinkage of gingival scar tissue resulting in the obliteration of the pockets is still in doubt.

—*G. H.*

374—Bouland, Dr. A.: **Pyorhées alvéolaires et radiography.** Pyorrhea alveolaris and radiography, in: Semaine Dentaire, 17: 663-668. 1935.

The author remarks upon the indispensability of radiography in the treatment of pyorrhea because of its value in determining the diagnosis and prognosis as well as following the results of treatment. Furthermore, the recalcification achieved by certain workers, particularly Hulin, will add further value to the X-ray as a means of controlling the results. Three case histories treated by the method of Kostritsky are presented.

—*G. H.*

375—Boyens, P. J., D. D. S.: **Safeguarding paradental health in dental reconstruction,** in: J. Calif. State D. Ass'n. 11: 11-12. 1935.

Dr. Boyens recognizes the importance of safeguarding the health of the paradentium especially in reconstruction work.

Shell crowns should not be used. Gingival margins of inlays should be smooth and well contoured. There should be no undue stress from partial dentures.

—*S. H. S.*

376—Buck, W. C., D. D. S.: **A rational concept of the treatment of periodontal lesions,** in: Minneapolis Dist. D. J., 18: 9-16. 1935.

Periodontal lesions may be grouped under three general divisions: 1. Gingivitis, 2. Pocket formation, 3. Diffuse alveolar atrophy.

Gingivitis, a mild inflammatory condition of the gum, is the precursor of pocket formation and must be recognized and treated early by home care, prophylaxis and removal of local irritating factors.

Pocket formation, called by Box "Periodontitis Simplex," by Gottlieb "Marginal Pyorrhea," by Kronfeld "Paradental Pyorrhea," comprises 80% of all pyorrhea cases. It is caused almost entirely by local irritating influences. The main characteristic is the presence of the pocket along the root. Treatment includes ultra violet therapy, direct electrical therapy, medicinal therapy, subgingival curettage, and surgery. Subgingival curettage is an attempt to secure re-attachment of tissues to tooth surface or at least such a tight adaptation as to render access by probing difficult, is unsatisfactory and of little practical value. The possibility of re-attachment is doubtful and epithelial proliferation rootwise is always likely to interfere with connective tissue re-attachment and cementum formation. The author prefers surgical excision of the pocket. The method preferred is gingivectomy following the undulating line of the gums.

Diffuse alveolar atrophy comprises about 10% of all pyorrhea cases and is caused almost entirely by systemic influences, metabolic, glandular, etc. It is not at present curable. Symptoms include the wasting of the alveolar process and the loosening of teeth. Treatment should be both systemic and local.

—*G. H.*

377—Chazelle, Ch.: **Evolution concomitante d'une paradentose pyodontoclasique et d'une maladie de Basedow.** Simultaneous evolution of a pyodontoclasic paradentosis and Basedow's disease, in: Semaine Dentaire, 17: 151-158. 1935.

The author reports the case of a young woman twenty-seven years of age who came to him for treatment of a suppurative periodontoclasia which had started nine

years before with the first symptom of hyperthyroidism. Investigation revealed that the patient's symptoms of psoriasis hyperthyroidism and paradentosis, sprang from an hepatic dysfunction. On the basis of this diagnosis of hepatic dysfunction causing psoriasis, hyperthyroidism and paradentosis the disintoxication cure of Gelpa was instituted. The psoriasis completely disappeared as did the respiratory spasms, tachycardia and other hyperthyroid symptoms. Local treatment consisted of a sclerosing solution of 20% quinine urea and a desensitization to toxins by the injection of sodium and magnesium hyposulfite.

—*G. H.*

378—Cremona, Robert-Guy: **L'importance de la cure des intoxication associée au traitement local, chez paradentosiques.** Disintoxication associated with local treatment in paradentosis, in: Semaine Dentaire, 17: 717-722. 1935.

The author presents the case of a woman, 55 years of age, suffering from a severe paradentosis. The patient's symptoms included digestive troubles, migraine, vertigo, loss of equilibrium and a browned skin. The paradentosis was considered as a tegumentary manifestation of arthritis due to an hepatic insufficiency. A state of humoral instability existed. Histologically this was shown by the presence in the gums of cells characteristic of anaphylactic shock, i.e. monocytes and eosinophiles.

A desensibilization to azotic residues is a necessary therapeutic for such a condition. The administration of sodium and magnesium hyposulfite according to the disintoxication cure of Gelpa was instituted. Local treatment consisted of intravenous injection of quinine urea according to the sclerosis method of Hulin. The local and general results were satisfactory.

Local treatment should be associated with the general treatment of the disintoxication. Complete cure can not be affected without treatment of the primary cause.

—*G. H.*

379—Dejou, G.: **Traitement des états pyorrhéique par le soufre colloidal.** Colloidal sulphur and the treatment of pyorrhetic conditions. Abst., in: Revue de Stomat., 37(1): 59. 1935.

Dejou maintains that periodontoclasia is not a purely local condition, but that it attacks the tissue after it has been weakened and prepared by a previous or existing systemic condition. It is associated most often with arthritis.

Robin has shown that in arthritic conditions there is a break down and loss of sulphur compounds.

The technique employed by the author is an injection of 1 to 2 cc of colloidal sulphur into the tissues. Some is put into the interproximal spaces, around the necks of the teeth and in the labial and buccal mucobuccal folds.

The sulphur acts by:

1. Its antiseptic power.

2. Stimulating general nutrition of the tissues.

3. Controlling the arthritic condition.

—*S. H. S.*

380—De Libouton, G.: **L'ozone en therapeutique bucco-dentaire.** Ozone in bucco-dental therapy, in: Semaine Dentaire, 17: 869-872. 1935. Abst., in: D. Cosmos, 915. 1935.

In granulomas and paradentosis ozone is used because, (1) of its bactericidal and cytophilic actions, which are very intense; (2) it has no inflammatory or pain reaction; (3) the treatment is very rapid; (4) it intensifies the reparative process; (5) there are no contraindications to its use.

E. A. Fisch of Zurich has lately written at length regarding his experimentations with ozone. In ozone (03) we have one molecule containing three atoms of oxygen with a density one and a half that of oxygen: 1.6584. It has a characteristic peculiar odor of fresh apples, which even in very small quantities is noticeable. It is obtained pure, except in the liquid state, and has an indigo-blue color; it boils at 100° C. It is obtained in a Berthelot apparatus by means of electric effluviums.

For its use in dentistry an apparatus is necessary, which, according to Fisch, is not at all complicated, being automatic, and regulating the amount and length of time of application. Just the same as in all root canal therapy, after its introduction into the root canal it has to be sealed up.

Fisch in his experimental work in the maxillary region on the cadaver, has found

that in osseous applications the penetration of the gas into the bony tissue is very great. No bacteria are resistant to its action, and all forms of organisms and their spores are destroyed in from five to thirty minutes.

Its application is painless and it is recommended by Libouton for all forms of suppurating states of the pulp and of the pericemental region, as also in paradentosis.

381—Dement, Robert L., D. D. S.: **Importance of early recognition and treatment of periodontoclasia,** in: J. Am. Dent. A., 22: 1307-1310. Aug., 1935.

It is important to recognize periodontoclasia in its incipiency and to treat it early. In this way the progress of the disease is halted and the prognosis is more favorable. Routine examination of all mouths should include: (1) Complete set of roentgenograms; (2) Vitality tests of all suspicious teeth; (3) Clinical examination along with roentgenograms; (4) Case history; (5) Prognosis.

—*S. H. S.*

382—Editorial: **"Once More,"** in: J. Am. Dent. A., 22: 304-306. 1935.

The editorial brings home the travesty of the statement that pyorrhea can not be cured. There is no need for the loss of so many teeth from pyorrhea.

"To make the offhand statement that pyorrhea cannot be cured and let it go at that, is a travesty on fact and an acknowledgement of gross inefficiency on the part of the one who makes it." . . .

"Failure on the part of the practitioner to co-operate with the patient to the limit of his ability in any case of pyorrhea is a definite reflection on the intent and ethical standard of the one who so fails. Patients who wish to save their teeth are entitled to receive the benefit of the best skill available, . . ."

"But the one thing most needed is a more sincere effort on the part of the profession to combat the trouble, and a more pronounced change of attitude toward the whole situation. It should no longer be considered honest for any dentist to foster the idea that pyorrhea cannot be cured. Under proper conditions it can be cured, and the needless sacrifice of the natural teeth should be stopped. The undue laudation of full dentures has influenced many patients to neglect their natural organs and resort to artificial substitutes, substitutes which they learn too late do not belie their name—they are nothing but substitutes. . . . Even if the cure of pyorrhea cannot be considered 100 per cent successful, neither can it be contended that the use of artificial dentures is ever 100 per cent satisfactory."

—*Extracts.*

383—Fantus, Bernard: **Therapy of fusospirochetosis (fusospirillosis).** A summary of views of various members of attending staff of Cook County Hospital, Chicago, in: J. A. M. A., 104(9): 741. March 2, 1935.

Two special localizations of Vincent's organisms, that is oro-pharyngeal and broncho-pulmonary, are discussed. Correct diagnosis is important as treatment is almost specific. Disease must be differentiated from diphtheria, syphilis, scurvy, agranulopenic angina and mercurial stomatitis. Finding of Vincent's symbiosis alone is not diagnostic, other characteristic manifestations of the disease are necessary.

Among prophylactic measures, careful mouth hygiene and dental asepsis are suggested. Soap is desirable in dentifrices. Improvement of general resistance is important. In treatment, the regulation oxydizing agents are recommended, hydrogen peroxide, sodium perborate, and chromium trioxide in conjunction with hydrogen peroxide; and as a spirillicide, neoarsphenamine locally and, in obstinate cases, intravenously. In general therapy, cathartics and analgesics may be prescribed. Smoking is contra-indicated and a vitamin-rich diet necessary. It is advised that surgical work, even scaling and polishing teeth, should be deferred during acute symptoms. In broncho-pulmonary fusospirochetosis, thorough oral hygiene should be instituted in addition to suitable systemic therapy.

384—Ginestet, Felix: **Traitement de la stomatite ulcéro-membraneuse par le cyanure de mercure en injections intra-musculaires.** Treatment of ulcero-membranous stomatitis by intra-muscular injections of cyanides of mercury, in: Revue de Stomat., 37(10): 641-644. 1935.

The author presents two cases diagnosed as ulcero-membranous stomatitis in which

the intra-muscular injection of mercuric cyanide was attendant with successful results. After about 7 or 8 injections the condition cleared up. The injections were made deep into the buttocks. There were no untoward local or general reactions observed.

The drug is of value in all ulcero-membranous conditions, unless it is contra-indicated by such conditions as nephritis, T. B., acute heart conditions, etc.

—*S. H. S.*

385—Gladwin, H. C., D. D. S.: **High heat as applied to the oral cavity,** in: D. Cosmos, 77: 884-887. 1935.

The author applies the Elliott principle of high heat treatment to the reduction of oral inflammation and infection. Rubber applicators fit over the upper and lower teeth and gums. Water at the desired temperature is circulated through the applicators. Nature in combating inflammation increases the blood supply to the pathological area. Heat does similarly by inducing a hyperemia, leukocytosis, accelerated resorption, increased lymph circulation, and speedier tissue regeneration. Temperatures from 115-122° F. are employed from twenty to forty minutes. The treatment is used by the author after prophylactic treatment as a means of stimulating the tissues. In cases of Vincent's infection two or three heat treatments prior to instrumentation shorten the acute stage of the disease. After instrumentation daily treatments at temperatures of 115-122° F. for thirty minutes are helpful in speeding recovery. In treatment of dry sockets daily Elliott heat treatments induce a blood clot with alleviation of pain.

—*G. H.*

386—Hardwick, H. L., L. D. S.: **Gingivectomy,** in: British D. J., 59: 400. 1935.

Report of a demonstration explaining the methods of treating paradental disease as performed in the Paradental Department of the Royal Dental Hospital. The report follows: "Particular reference was made to gingivectomy and the technic of the operation was shown on a model with plasticene gum. Thus the exact angle of the incisions could be clearly seen. After-treatment consisted of the cleansing of the interdental spaces by the patient with wooden toothpicks. It was emphasized that strict attention to great detail, with conscientious cooperation by the patient were essential factors in determining the success of the treatment."

—*G. H.*

387—Hartzell, T. B., D. M. D.: **The story of the origin and development of periodontology,** in: J. Am. Dent. A., 22: 1899-1908. 1935.

Dr. J. M. Riggs, of Connecticut, who first directed attention towards pyorrhea in the U. S., treated the disease by subgingival curettage. His treatment was mechanical and made no reference to the etiological role of bacteria in pyorrhea. Inasmuch as Hartzell believes that periodontoclasia is a germ ferment disease and periodontal and alveolar bone destruction are due to the action of purulent organisms he feels that little has been accomplished in therapeutics of this disease since Rigg's time. Root cleansing alone is of no permanent value in treatment since it produces only temporary results. Cases with deep pockets will show a recurrence if treated by curettage alone.

Hartzell points out the danger of injuring the delicate cementum during root planing and thus implanting future infection. Fletcher's method of surgically removing periodontal pockets and diseased bone around such pockets was a great step forward. Barnes, Fones, Smith, and Charters, added to the progress in treatment by teaching proper toothbrushing methods and interdental stimulation as a means of keeping teeth and gums free from bacterial growths.

Bacteria possess the power of penetrating gum tissue, and with their role as foci of infection in connection with infected apices a treatment of more than root curettage is called for. The neutralization and detoxification of the products of bacterial growth is of prime importance in suppressing periodontal inflammation. It is not the bacteria themselves but their toxins which destroy tissue. Larson's discovery of a harmless agent to neutralize these bacterial ferments is one of the most useful aids in the treatment of periodontal disease. —*G. H.*

388—Hess, W.: **Einige Betrachtungen zur Arbeit, "Diagnose und Therapie der Stoffwechselstörungen bei paradentose,"** in: Paradentium, 7: 117-121. 1935.

A consideration of the article by Dr. F. Bircher-Rey on the diagnosis and therapy of paradentosis in relation to disturbances in metabolism.

The author after considering the five cases offered by Bircher-Rey as evidence for improving and healing paradentosis by diet comes to the conclusion that the investigator's method is not scientific enough to be conclusive. He offers suggestions whereby a true healing or improvement can be indisputably established.

1) A true condition of paradentosis should be established by X-ray at a definitely noted angulation before treatment begins.

2) After the treatment with an established diet, X-rays should again be taken at the previously noted angulation so as to do away with doubts or optical delusions.

3) The X-rays should be stereoscopic where possible.

4) The scientific evidence of a true healing diet can only be determined by pathoanatomical observations on cadavers.

5) An improvement of paradentosis by diet alone which takes a long period of time should be evidenced on X-rays as it goes along and the clinical symptoms should disappear.

—*N. K. B.*

389—Hogg, O. S. B.: **The co-operation of the patient with the dentist in the treatment of pyorrhea,** in: Dent. J. of Australia, 7: 291-293. May, 1935.

The author expresses the belief that a typewritten sheet given to the patient containing the causes of, and the home care for pyorrhea will go a long way toward interesting the patient and eliciting good results during treatment. He goes on to discuss exactly what this instruction sheet should contain.

The causes of pyorrhea are mentioned.

The patient's care consists of:

1) Maintaining a normal, or slightly higher than normal, calcium-phosphorous content in the diet through the ingestion of vegetables, egg-yolk and milk;
2) Roughage-massage of the gums attained by eating celery and raw fruits;
3) A diet rich in all vitamins—through dairy products and vegetables;
4) Sleeping with a closed mouth (mouth breathers invite bacterial growth on dried saliva when they sleep with mouth-open).
5) Cleaning the oral cavity thoroughly. The intelligent use of a brush and *paste.*
6) Swabbing of pockets with cotton pellets saturated with pure peroxide of hydrogen;
7) A saline mouth wash with vigorous mouth flushing.

—*S. E.*

390—Hulin, Ch.: **La gingivo-fibrose profonde et son complément ostéogénétique dans la traitement des paradentoses.** Deep gingival fibrosis and its osteogenetic complement in the treatment of paradentosis, in: L'-Odontologie, 73: 85-110. 1935.

The author traces the evolution of the treatment of gingival fibrosis from Fauchard to the present day. As early as 1728 Fauchard employed cautery as a therapeutic cure of pyorrhea.

The therapeutic criterion to be achieved is the formation of new fibrous tissue in the place of the destroyed paradentium.

Hulin considers salivary calculus to be formed of a framework of organic material, the most prominent of which is mucin. As a glycoproteid mucin has an affinity for Ca with which it forms an adsorption complex. Thus in salivary calculus and cartilage, the mucin of the former and the collagenous material of the latter adsorb Ca salts. The same principle may be applied to bone. Osseine which is a collagenous substance acts as a depot for calcium deposits.

The process of fibrosis leads to the disappearance of the polymorphs, hypervascularity and produces a condition which is favorable towards bone formation. Thus, the fibrosing of pyorrhetic pockets is a preliminary step in the regeneration of alveolar bone. In addition the Ca and PO_4 ion concentration must be maintained. Moreover, certain proteins in excess will prevent the Ca deposition. These should be avoided in the diet. Thus, hepatic disorders must be treated before bone regeneration is possible.

Fibrosis is obtained by the injection of

a solution containing basic quinine chlorhydrate, urethane, formaldehyde and methylene blue. The osteogenetic treatment consists of injecting a solution of a homologue of osseine (to constitute the organic framework of the bone), some mucins, some glycogens and a tissue diastase into the fibrosed areas. The work is, however, in the preliminary stage. Only one case so treated is cited.

—*G. H.*

391—Hulin, Charles: **Treatment of pyorrhea by means of osteogenic fibrosis,** in: L'Odontologie. Abst., in: British D. J., 59: 391. Feb., 1935.

Charles Hulin, in a study of periodontal disease, believes that regeneration of bone can take place by means of fibrosis. Sclerosis is defined as a process resulting from local and systemic tissue-cell interaction. Inflammatory processes produce this process, which is one of several methods of repair. Hulin prefers the term "fibrosis" in describing the intensive fibrous transformation of the peridental tissues obtained by injecting certain liquids into diseased tissue. There is a multiplication of epithelial cells which invade the deeper connective tissue layers with proliferation of leucocytes hindering the repair of bone. The introduction of this liquid stimulates fibrous tissue formation and inhibits cell proliferation. Fibrous tissue *can* become osseous by metaplasia; this tendency to form fibrous tissue and consequent removal of polynuclear cells, an important check to bone repair, is removed; fibrosis restricts the blood supply and ischemia, so that calcification can take place at a distance from the vessels; fibrous tissue is formed in an unequal and irregular manner, leaving spaces filled with connective tissue into which fibrinoid matter can filter and become liable to calcification, that is, form a center of crystalization.

If the mobility of the tooth remains within physiological limits or disappears with fibrosis there will be no disturbance of the intercellular colloidal elements, which hinders calcific precipitation; the lesion being a bony one, the repair taking place in proximity to bone induces local changes which support the assertion that calcification and absorption are always present. The presence of bone has a powerful effect; according to Ollier, "tissues which will not ossify when irritated by themselves are however susceptible of ossification when stimulated concurrently with bone in their neighborhood." The treatment with fibrosis bears this out, for new bone is formed from the salts of the bone already present; this so-called "healing fibrosis" produces a medium which can be ossified. If the calcific material is partly formed from the neighboring bone the blood plasma will supply the phospho-carbonates of calcium, in the form of complex colloids, which precipitate readily in the meshes of the connective tissue from the newly formed fibrous network.

Hulin injects into the gum the following solution:

1. Basic hydrochloride of quinine—3 gr.
2. Urethane—17 gr.
3. Formaldehyde—1 gr.
4. Methylene blue—0.2 gr.
5. Water—100 gr.

The underlying idea is the introduction of a substance homologous to osseine into tissues which have been previously made fibrous. Hulin found that the mucus of snail, which contains calcium phosphate, to which was added a diastase extracted from vesical membrane, glycogen and gelatin, injected into tissue rendered fibrous, acts as an osteogenic agent and without any extraneous calcium. The solution contains:

1. Vesical diastase—.001.
2. Glycogen—0.02.
3. Mucin—0.10.
4. Gelatin—0.5.
5. Physiological solution—2 cc.

All together sterilized by exposure to ultra-violet rays.

Severe pyorrhea treated in this fashion shows disappearance of pus and teeth firmly fixed in firm alveoli.

392—Huttinger, Dr.: **Le traitement de la pyorrhée et l'hygiene buccale.** Treatment of pyorrhea and local hygiene, in: Semaine Dentaire, 17: 789-795. 1935.

Bacteria are merely secondary invaders in pyorrhea. The primary cause is a diminution of tissue tonicity and resistance. When pyorrhea affects isolated teeth only such cases may be attributed to local causes, as

for example loss of antagonistic teeth. There is, however, a generalized type of pyorrhea which attacks all the teeth. The cause in such cases is systemic in nature. The first symptom is recession of the gingiva exposing them to infection. It is this type of pyorrhea about which so much is written but so little is actually known.

The true constitutional pyorrhea is the local manifestation of a lowered body resistance and will be as difficult to cure as it will be to rehabilitate the system from the condition responsible for the debilitation. Local curettage and massage will reduce the infection but does not result in a complete cure unless the patient takes extreme pains with oral hygiene. Toothbrushing will not suffice. Special care must be taken in cleansing interdental spaces, exposed roots, etc. The author recommends the use of fine syringe placed underneath the gingiva in inaccessible areas.

—*G. H.*

393—Ivanoff, Dr.: **L'ozone in therapeutique dentaire.** Ozone in dental therapeutics, in: Semaine Dentaire, 17: 29-30. 1935.

Pure ozone is a bactericide, oxidant, tonic and sedative. Prof. Marion has shown that a current of ozone passed through a staphylococcus culture for six minutes inhibits the growth of the organisms. The gas, passed into a suppurating wound causes lysis of the microörganisms (Dr. Mathis, 1931).

While not claiming it to be a universal panacea, Dr. Ivanoff advocates the use of ozone in cases of gingivitis. The conditions usually clear up with about six to eight applications of the treatment. The technic employed involves the injection of the gas into the gingival tissue with a hypodermic syringe. In the last treatment the gas is applied to the affected part under pressure.

The use of ozone recalls to mind Dr. Dunlop's oxygen therapy.

—*S. H. S.*

394—Lajugie, Mme.: **Influence de la stabilisation mecanique sur la regeneration osseuse chez la paradentosiques.** Influence of mechanical stabilization on osseous regeneration in paradentosis, in: Semaine Dentaire, 17: 313-318. 1935.

In order to fully illustrate the value of mechanical stabilization in bone regeneration the author first discusses the influence of mechanical disequilibrium. She considers paradentosis as due to a humoral instability, a state of anaphylactic sensitivity and, therefore, mechanical disequilibrium is only an aggravating cause in the evolution of the disease. Of great importance as a traumatizing factor are tangential forces since the teeth can withstand such forces less readily than those of a vertical nature.

Mechanical irritation results in a chronic inflammation with blood stasis and an excess of carbon dioxide. This excess carbon dioxide causes a local cellular asphyxia. In order that gingival fibrosis may occur, the intercellular albumin must be oxidized to collagen. Therefore, mechanical stabilization is essential to the success of gingival fibrosis in that instability will cause inflammation with the preceding results. Moreover, calcification of the alveolar bone will not readily occur if mobility is present. The connective tissue framework of the area of deposition must be persistent and immobile. The mechanical stabilization should be sufficient to favor bone reparation and should be complementary and constitute the final phase of the treatment.

—*G. H.*

395—Leclerq: **Contribution a l'étude des stomatites. Revue clinique et traitement par les cobrants d'acridine** (These de Paris, 1935), Contribution to the study of stomatitis. Clinical revue and treatment by the acridine dyes. Review in: Revue de Stomat, 37(11): 774. 1935.

The author recommends the use of the acridine dyes in the treatment of stomatitis. He bases his rationale on the assumption that in all cases, infection is either primary or superimposed. From the preventive standpoint, the treatment is very valuable.

The advantages are that the treatment is simple, the patient can continue in his regular work; there are no contra-indications; in medicamentous stomatitis systemic treatment (syphilis) in progress need not be interrupted.

—*S. H. S.*

396—Leonard, H. J., D. D. S.: **Periodontia treatment for maximum attachment,** in: J. Am. Dent. A., 22: 602-607. 1935.

Simple periodontoclasia is a chronic inflammation which starts at the base of the gingival sulcus and gradually causes destruction of the portion of the pericementum and alveolar process in the advancing inflammatory area. The root is consequently denuded and a pocket is formed, lined with a weak and degenerated epithelium. The exposed cementum surface is full of organic putrefying fibre ends and, in most cases, serumal calculus. The surrounding tissues are inflamed and infected with Streptococcus viridans.

The best method of treatment in such conditions involves the careful curettage of the root surfaces to free them of all subgingival calculus and roughness, and to shave away the thin outer layer of cementum containing the fibre-ends. Occlusion is adjusted to eliminate trauma and food impaction. The treatment must be followed up by adequate home care, designed to keep the teeth clean and the gums massaged. The massage consists of a series of pressures and releases, calculated to keep up an active flow of blood through the tissues.

As to whether reattachment may occur, two cases are cited in which the evidence is strongly in favor of the possibility. Laboratory data in this connection is very difficult to obtain. Theoretically it is conceivable that a reattachment may take place inasmuch as injured cementum and pericementum can heal normally if there is no opening to the surface. With the above treatment the pocket is kept constantly filled with blood clots so that the base is not exposed to the irritating environment of the oral cavity.

—S. *H.* S.

397—Loisier, Pierre: **Essai sur l'histoire des paradentoses et sur leur thérapeutiques.** History of the paradentoses and their therapeutics, in: L'Odontologie, 75: 698-711. 1935.

The author presents a history of the development of the various ideas concerning paradentosis, tracing them through the centuries and showing how each scientific decade added its bit to the final present day theories. Caries and pyorrhea existed in the days of the Pharoahs but it was not until the sixteenth century that a clinical description of pyorrhea was adequately presented by Paré. From that time on the author presents the dates and names of the various men who contributed to the clinical and therapeutic knowledge of pyorrhea, as well as giving their contributions.

—*G. H.*

398—Lose, D. D. S., and Foure, D. D. S.: **Observations on periodontoclasia with special reference to the use of autogenous vaccines and surgical intervention,** in: D. Cosmos, 77: 868-877. 1935.

The experiments were performed on individuals free from complicating diseases such as nephritis, diabetes, tuberculosis, frequent colds, loss of weight, etc. An initial curettement and vaccine treatment was given all patients who were then divided into two groups. Group 1 received subgingival curettage on the right side every thirty days. Group 2 had a gingivectomy performed on the right side only. The left side of the mouth in both groups was left alone after the initial curettement.

The authors found that the reaction to the vaccine was varied, usually resulting in general mouth soreness and in some cases stiffening of the joints. Vaccine treatment is an adjuvant to curettement and aids the tissues in responding to mechanical treatment. The rapidity of the improvements noted are attributed to the use of the vaccine. Mechanical irritation is the primary local cause of periodontoclasia and the use of autogenous vaccines is only an adjunct to speed up refractory cases. The technic of preparing and administering autogenous vaccines is described.

In group 1 it is interesting to note little difference between the two sides of the mouth, one side receiving only an initial curettement while the other received a periodical scaling once a month. This might show that an initial curettement might produce an immunity and is as effective as periodical curettement.

In group 2 gingivectomy resulted in quicker relief but with more destruction than the conservatively treated side. Due to the final appearance of the mouth it is deemed inadvisable to resort to radical surgical elimination of the pockets. The authors observed no bone regeneration in all cases one year after treatment. The reattachment seems to be a soft tissue one.

—*G. H.*

399—Merritt, Arthur H., D. D. S.: **Prevention from the viewpoint of the periodontist,** in: D. Outlook, 22: 328. August, 1935.

Any attempt at preventive dentistry must include periodontia. The maintenance of healthy supporting structures is vital to all restorative work. Prevention must precede remedy. Real progress in dental care by the general practitioner can only be made by his prevention of periodontal conditions.

The local and systemic causes for periodontal diseases are listed. The author believes that a control of local factors practically removes all periodontal pathological tendencies.

The preventive measures are:

1. Develop a "clean mouth." A typical method is outlined.
2. Teach patient how to maintain such cleanliness.

Treatment is to be instituted by:

1. Apoxesis.
2. Polishing (iodine and porte polisher).
3. Dental restorations. (Anatomy and functional occlusion regulated.)

Home methods to be used are:

1. Proper toothbrushing methods.
2. Waxed floss.
3. Massage.

Progress having been made, the patient must be seen two to four times a year for checkup.

400—Merritt, A. H., D. D. S.: **Vincent's infection and periodontoclasia; the secret of successful treatment,** in: J. N. Y. Acad. Dentistry, 2: 41-50. June, 1935.

The author's own summary of his article follows:

"Vincent's infections. Clinically there are two types of Vincent's infection; acute and subacute. The weight of evidence at present points to the fusospirochetal organisms as the probable cause. There are two important predisposing factors in Vincent's-oral sepsis and excessive smoking. Young adults are most susceptible. Treatment consists in the establishment of good hygiene and in the use of oxygen liberating drugs. The secret of success in treatment is a careful prophylactic treatment at the first visit plus the liberal use of oxidizing agents.

Periodontoclasia. This is characterized by the formation of pockets through the loss of alveolar bone. It may be caused by alteration in the acid-base balance, which may or may not be present, and by several local factors which are always present. Treatment includes, a) local operative procedures, b) balancing occlusion, c) systemic treatment, d) post-operative care. Four different operative procedures are outlined: subgingival curettage, flap operation, modified flap operation, and gum resection. Tooth grinding and the replacement of lost teeth are important factors in balancing occlusion. Systemic treatment includes an adjustment of the diet, blood examination and coöperation with the physician. Postoperative treatment includes instruction of the patient and prophylaxis. The secret of success in the treatment of periodontoclasia is early treatment.

—*G. H.*

401—Miller, I. S., D. D. S.: **Some of the clinical results of a standard periodontia procedure, illustrative of the principles involved,** in: D. Cosmos; 77(4): 365-369. April, 1935.

For the most effective treatment of gum diseases, the author presents a plan of standard periodontia procedure. The principles underlying the procedure are discussed under six divisions. (1) Knowledge of the normal anatomy and physiology of the parts. (2) All causes in each case must be known and removed, if possible. (3) The etiological factors are usually local, namely mechanical, chemical and bacterial. (4) The operator must believe that pyorrhea is a curable and preventable disease. (5) By the use of rational treatment it should be possible to retain the natural teeth through old age, in most cases. (6) The oral spirochetes and related anaerobes are the "most active destructive agents universally present in all cases of periodontal disease."

The use of quaiacol, 2 parts, and eugenol, 1 part, as a spirillicide at the chair is recommended.

The steps in treatment are outlined as follows: (1) Diagnosis, (2) Balancing occlusion, (3) Instrumentation, (4) Mouth hygiene and tissue stimulation, (5) Planning of necessary restorations, (6) Prophylactic treatment.

Dr. Miller claims that among other results of such a standard practice, he gets a

regeneration of the alveolar process about the affected teeth.

—*S. H. S.*

402—Morgan, Agnes F., et al: **Effect of diets on calcium and phosphorous metabolism in dogs.** Abs., in: J. Am. Dent. A., 22(2): 339-340, 1935. (Original article in U. of Calif. Publications in Physiol. 8:61. No. 7.)

This is a study of the effect of special diets on the bones, blood and excretions, teeth and gingivae. The experimental tests were made on young dogs.

Observations:

1. Alkali-fed dogs were usually larger at every age than comparable animals on other diets.

2. The bones of those given Vit. D (cod liver oil) were nearly normal, that of the dogs of vitamin D free diet were rachitic.

3. The vitamin D free diet produced severe lesions whether the diet was acid or alkaline.

4. The low calcium group showed the severest lesions of all.

It is concluded that paradentosis in varying degrees of severity as well as renal damage may result from long continued use of alkaline diets either with or without vitamin D and normal calcium-phosphorous ratio and content.

—*S. H. S.*

403—Muller-Stade: Question: **Bleeding of gingiva,** in: Deutsche Zahnärtz. Wochensch., 38: 236. March, 1935.

Question: A nine year old girl suffers from intense bleeding of the gingiva on brushing of the teeth. Mouth condition: has gold fillings, no amalgams, hypoplasia, gingiva is red and swollen. Weeks of treatment consisting of cauterizations, mouth-rinsings, massage, atomizers, etc. What is treatment?

Answer: The disposition towards the unusual amount of bleeding points to an endocrine disturbance. A routine of brushing with iodized potassium chlorate with a soft tooth brush and finger massage should be instituted. Since there is no objection to using strong solution of iodine in children who have not reached puberty, a strong one can be prescribed here.

—*N. K. B.*

404—Murphy, R. H.: **Classification of types of periodontia,** in: Florida D. J. Abst., in: D. Outlook, 22: 56. Jan., 1935.

Treatment:

The best service to be rendered a patient is a thorough *monthly* prophylaxis, with definite instructions as to home care. Home care should be checked on carefully. The presence of periodontal lesions requires X-rays without delay. If possible, the case should be handled with *deep scaling* under novocaine. Hypertrophy of the gingiva, weepy lifeless bleeding call for *gum resection.* The flap operation should be carried out where teeth are loose with suppurating pockets approaching the root ends. This is especially true in the posterior region. Conservative surgery should be used in the anterior of the mouth.

Methods will vary slightly to suit individual cases.

405—Norcy, Dr.: **Le calcium et sa fixation dans les tissus dans la therapeutique régénératrice du paradentium.** Calcium and its fixation in the tissues in the therapeutic regeneration of the paradentium, in: Semaine Dentaire, 17: 465-486. 1935.

The author presents the theories of the several French schools of thought on the etiology of paradentosis. He believes that Hulin's ideas seem to approach the nearest to the truth although the absolute solution is not yet at hand. The début of the disease is the penetration of the gingival epithelium by bacteria, depending on the two factors of tissue resistance and virulence of the microörganisms. However without an ionic disequilibrium the disease would be a simple gingivitis and not a paradentosis. There must exist a certain local anaphylaxis produced by an antigenous albumen or microbe. Such a sensibilization causes asthma in the level of the bronchi, in the blood urticaria, etc.

The various roles of calcium in the body are briefly discussed. Included are its part in the transmission of nervous and muscular impulses, muscular contraction, the activation of pancreatic juice, circulation, etc. An interesting note is made on the work of Gordon and Jorg who suggest that dentinal sensitivity is transmitted to the odontoblasts by calcium ions.

The level of blood calcium varies little and is distributed equally between plasma and red cells. The types of blood calcium (30% colloidal, 60% ionizable, 10% diffusible but non-ionized) are reviewed. The level of the blood calcium is not, however, an indication of the calcification or decalcification of other tissues as is evidenced by the lack of change in blood calcium in Paget's disease, acromegaly, hyperostosis, osteoporesis, osteomalacia, osteitis, etc. This indicates that the level of blood calcium is fixed by the reserve in the bony tissues and is regulated by the parathyroids. After discussing the fixation of calcium in the tissues the author turns to the use of calcium in therapeutics.

The gingivo-dentary region is an autonomous one and differs from other skeletal tissues as is evidenced by the resistance of carious teeth and paradentosis to ultraviolet rays which bring about a rapid recalcification in other parts of the skeleton.

The treatment of paradentosis by deep gingival sclerosis is brought about by intragingival injections of lipoids to provide a fibrolligenous base and calcium chloride to stimulate cellular growth and osteogenesis. The author prefers the inorganic calcium salts to the organic (lactates, gluconate). He varies the dose with the severity of the case.

—*G. H.*

406—Pike, Jay N.: **Malocclusions in their relation to periodontal disease,** in: Minneapolis D. J., 18: 17. March, 1935.

Malocclusions, with their relationship to periodontal disease, are found usually in adults,—but these conditions begin in children.

In cases where there is a questionable cause-effect relationship between malocclusion and periodontal disturbances, it seems that the malocclusion is not a basic cause to the latter condition.

The Angle and Dewey definitions of malocclusion are given.

The periodontist limits his treatments of malocclusion to traumatic occlusion and works only on individual sites. The orthodontist, in his work, includes large parts or even the entire mouth in his concept of malocclusion. Like the latter, the periodontist should, in order to give his patients a broader and completer service, take the latter point of view.

Grinding is one method used by both periodontist and orthodontist. Teeth which have moved or been moved into a new alignment do not have "normal wear" in the correct sites. When a bite is opened bicuspids and molars must be ground occlusally to new occlusal surface forms to compensate for lateral movements.

The author describes several other techniques for obtaining normal functional occlusion. These techniques are of such a nature that they reduce periodontal involvement to a minimum; thereby "traumatic occlusion" disappears at the same time. These methods include use of splints, cement blocks, and grinding of these splints or blocks in order to release locked bites, help jaw movement, establish normal centric relation and lateral movement, and restore lateral balance. In any case, the method used must be used carefully and judiciously.

The attainment of functional occlusion will give better results to the orthodontist and will serve to control traumatic occlusion for the periodontist.

407—Sanders, A. E., D. D. S.: **Pyorrhea surgery for the general practitioner,** in: D. Digest, 41: 168-170. May, 1935.

The author has devised a set of four instruments with double ends which will reach and remove all portions of the gums neatly and cleanly. He believes that it is not necessary to cut down to good bone and gum tissue, especially bone, in order to eradicate pyorrhea. A complete mouth gingivectomy should not be done at one time. Such an operation would expose the blood stream to a great amount of infection. Gingivectomy is desirable because it removes the pockets and enables one to get a good view of the roots for scaling. Subgingival curettage is often a failure because of incomplete removal of deposit. Proper home care constitutes an important part of the treatment. Gingivectomy is an easy operation and can be performed by the average general practitioner.

—*G. H.*

408—Scherer, W. H., D. D. S.: **Dunlop method of treating periodontal lesions,** in: Texas D. J., 53(1): 2-9. 1935.

The author outlines a method of treatment for periodontal lesions as advanced by Dr. Dunlop. The Dunlop method is comprised of three parts. The first is the introduction of oxygen into the subgingival tissues. The theory is that the introduction of oxygen raises the alkalinity and thus favors phagocytosis, increases oxygen tension, thus hastening metabolism of the cells, and raises the oxidation-reduction potential.

The second component is a paste, containing among other things, glycerin and boric acid.

The third component is a surgical wax, which is pressed down into the pockets. This causes an atrophy of the affected tissue without loss of healthy tissue.

Mention is made of Dr. Box's application of the Guaiac test for the detection of minute gingival pus. A mildly positive test does not denote infection of the gingival tissue, although a strongly positive test is considered fairly reliable as to the presence of infection.

—*S. H. S.*

409—Sebald, H. A., D. D. S.: **Pyorrhea,** (Correspondence), in: J. Am. Dent. A., 22: 719. April, 1935.

The writer commends the editor for an editorial on *pyorrhea* in February issue and offers the following treatment for pyorrhea:

1. Pyorrhea should be prevented by good dental work, regular prophylactic care, correct home care. Coöperation is essential.

2. Pyorrhea in early stages *can* be cured by the dentist.

3. Persistent home care, regular reexaminations, and prophylaxis maintain cured pyorrhea.

4. Instrumentation—and not drugs cure pyorrhea. Strong drugs are absolutely contraindicated.

5. Surgical treatment is most logical—but as generally described, too radical.

a. Gum should be trimmed; laid back; inner surface thoroughly curetted.

b. Bone under pockets should *not* be curetted.

6. The treatment includes apoxesis, correction of occlusion and faulty dental work, and a general physical check-up.

7. Diet should be corrected and reënforced with vitamins A and D, calcium and phosphorous.

8. Dentist should teach *proper* brushing, flossing and interdental-rubber stimulation.

9. A four-week post-graduate course is necessary for proper approach and technique.

10. Pyorrhea treatment is a real service—and requires more study by the practitioner to enable him to give proper treatment to this prevalent condition.

—*Abstract*—*S. E.*

410—Silverman, M. M., D. D. S.: **Flap operation for the preservation of the gingival crevice,** in: D. Cosmos, 77: 987. October, 1935.

The author presents a modified procedure for the incision in the "flap operation," which conforms to recent findings by research workers on the histological structure of the gingival attachment.

The various flap operations now in use are discussed. They are:

1. Radical technique—with maximum bone exposure and destruction of the gingival attachment.

2. Semi-circular or conservative type—with lack of sufficient bone exposure.

3. Curtain type—with sufficient exposure but curtain interference during operative procedure.

The type used by the author is a combination of 1 and 2. With photographs, the author demonstrates how his method gives maximum bone exposure and at the same time preserves the gingival attachment, thereby retaining nature's resistance to gingival infection. This method is applicable in operating on cysts, roots, foreign bodies, residual infections, impacted teeth, etc.

411—Silverman, M., D. D. S.: **Pain,** in: D. Cosmos, 77(12): 1211. December, 1935.

The author makes a plea for more rational therapeusis in oral conditions, both for the alleviation of pain and the removal of existing oral pathology. After giving an historical background to the use of drugs, he discusses the following conditions, and those drugs which ought to be used to correct these conditions:

1) Pain of operative procedures. 2) Dry

socket. 3) Post operative pain. 4) Pulpitis. 5) Perapical abscess. 6) Facial neuralgia. 7) Pericoronitis, of lower third molar. 8) Sinusitis. 9) Carcinomia.

The author feels that Vincent's infection gets its primary foothold, "only in dirty mouths." Sedation is to be obtained through the use of amidopyrine, five gr. every three to four hours. Hydrogen peroxide as a mouthwash every two hours, tablespoonful to one-half glass of water; rose-aniline dyes applied daily at the chair. Saline cathartics, plenty of row vegetables, orange juice and water.

Four types of drugs exist for treating Vincent's. They are cauterizing agents, oxidizing agents, germicidal dyes, and spirocheticides.

Cauterants are considered self-limiting in action and cause needless destruction of tissue (ex: chromic acid 10%).

The oxidizing agents yield good results (ex: sodium perborate or hydrogen peroxide).

The dyes are used effectively at the chair (ex: gentian violet or brilliant green).

By the use of the spirocheticides (arsenicals and bismuth) Vincent's infection is corrected in less time, with less permanent damage to the tissues, with pain being relieved within from one to two hours after the first application. Administration is intravenous as well as topical.

He concludes by advising practitioners when in doubt to write to the Council on Dental Therapeutics of the A. D. A. for information about any drug before prescribing it.

412—Smith, T. S., D. D. S.: **Constructive treatment of diseased paradental tissues,** in: J. Am. Dent. A., 22: 1477-1491. Sept., 1935.

Normally the epithelium of the gingiva is continuous with that of the enamel of the tooth. As the tooth erupts the attachment moves farther cervically. This structure serves to protect the underlying tissues from excursions of food and other irritants.

Correct treatment of paradental pockets is gentle curettage of the roots to remove all debris. The last act is to start a slight hemorrhage in the bottom of the wound, and to allow it to coagulate without any interference. The blood clot thus formed seals the wound and becomes organized into connective tissue. The blood also helps by virtue of its bactericidal properties. The epithelium on the tissue side of the pocket should be removed either surgically or chemically so that there will be open surfaces for healing. Usually the gentle action of a probe is enough to remove the thin and poorly organized epithelial covering and expose the connective tissue.

Photomicrographs are presented to show that the cementum can receive nutriment from the pulp as well as from the peridental membrane.

The cases presented are from the year 1903 with follow-up examinations for almost 30 years, showing "complete obliteration of deep, septic, paradental pockets."

—*S. H. S.*

413—Stillman, Paul R., D. D. S.: **An opinion as to the present status of dental hygiene,** in: D. Cosmos, 77: 1177-1186. 1935.

The author concerns himself with the biological approach to dentistry. Hygiene, anatomy, physiology, and pathology compose the four basic biological medical sciences. Dentistry's knowledge of these basic sciences should be essentially the same as medicine excepting for dentistry's anatomical limitations.

The biological philosophy has changed dentistry from the filling, cleaning, adjusting or extraction of teeth, and the providing of artificial dentures, to a "branch of the healing art, co-equal to a specialty of medicine." Dental hygiene, however, should not be confused with sanitation which can not be classed as a biological science. Though oral cleanliness is an important item in the oral health picture, it is a well known fact that clean mouths are not necessarily healthy ones. Dental hygiene delves further than the mere technique of cleaning the teeth. It seeks the etiology of the condition, the biological source. Moreover, the source being biological, so must the remedy be.

And, therefore, we have a change from technology to biology, from cleaning and examination, to diagnosis and a search for the source of uncleanliness. The science of dental hygiene must be incomplete, however, until the further development of the science of dental physiology which is far

behind dental anatomy and oral pathology.

In analyzing his own methods in restoring mouth health the author classifies them under three headings: 1) the grinding of teeth in order to establish a functional or physiologic occlusion; 2) the cleaning of teeth; 3) the restoration of tissue tone and resistance by proper tooth brushing.

—*G. H.*

414—Stones, H. H., M. D., Ch. B., M. D. S., L. D. S.: **Parodontal diseases: methods of treatment, including surgical eradication of pockets,** in: D. Record, 55(8): 460-467. 1935.

Dr. Stones reviews the different methods in current use for the treatment of periodontoclasia.

There is a brief description of each of the following methods:

1) The local application of antiseptics. 2) Specific remedies against the various microörganisms found in the pockets. 3) Systemic treatment. 4) Eradication of pockets.

Under the last named treatment there are four methods of approach.

a) Subgingival curettage—The author believes that this is of value only in that it removes serumal calculus.

b) The Dunlop wax and oxygen technique—Tendency for relapse.

c) Chemical removal of pockets by caustic potash. The disadvantage here is that it is difficult to control.

d) Surgical treatment. Dr. Stones believes that this offers the most efficient method of eradicating the disease. He describes the technique in brief detail, including (a) examination of the case, (b) preoperative treatment, (c) operation, (d) post operative treatment.

—*S. H. S.*

415—Strean, L. P., D. D. S.: **The correlation of the practices of orthodontia and periodontia,** in: D. Cosmos, 77: 399-401. April, 1935.

This article advises a closer relationship between the orthodontist and the periodontist. Too often the orthodontist completes restoration of esthetics in centric occlusion without considering the restoration of functional occlusion. Teeth must look well and function efficiently.

Six postorthodontic cases are presented to prove this need. These cases demonstrate, in order:

1) traumatic occlusion in lateral excursions and protrusive occlusion.

2) periodontal disturbances (traumatic crescents) due to faulty functional occlusion.

3) migrations of the teeth due to faulty functional occlusion.

4) loss contact of the teeth and loss of alveolar support due to faulty functional occlusion.

—*S. E.*

416—Van Der Ghinst, J.: **Pyorrhée alvéolaire ou periodontoclasie.** Pyorrhea alveolaris or periodontoclasia. (Congres International de Stomatologie, Bologne, Italie, Avril, 1935). Abst. in: L'Odontologie, 75(11): 787. 1935.

Numerous studies have indicated that pyorrhea is essentially related to calcium metabolism. On this basis general treatment could be instituted if it were possible to modify the amount of calcium, free in the blood. The parathyroids play an important role in this.

Locally the role of the periodontist is essential. The concept of traumatic occlusion serves as a guide not only in treatment but also in diagnosis.

—*S. H. S.*

417—Vastine, A. B., D. D. S.: **Subgingival treatment in oral surgery,** in: J. Am. Dent. A., 22: 953. June, 1935.

The author has made a study of subgingival treatment for many years. He believes that, "subgingival treatment has a wide range of use, but it is distinctly valuable as the most effective method . . . for preoperative preparation."

It is a most effective method for treating retromolar areas and erupting teeth; for controlling non-specific osteomyelitis, grafted on deficiency diseases, such as avitaminosis; for relieving acute periapical infection, and distinguishing it from an end stage infection; for treating adenopathies which have their origin in gingival or follicular infections; for treating, or preparing for operation in maxillary sinusitis of dental origin periapical, follicular or gingival sources, for reducing some large intra-oral cysts before operating (thus simplifying op-

erative technic) or treatment of facial dermatitis (erythema, acne or grave cellulitis of the nose) of dental origin.

The method employed by the author consists of:

1. Smear-taking and checking — following treatments, with subgingival cups and microscopic investigations.

2. Notation of normal incubation periods and growth cycles of organisms present.

3. Record of pH of subgingival field of the mucoid secretion and of the saliva, (to check on lowered resistances due to increased organism activity or alterations of metabolism).

4. The use of specific bactericides and amebicides with subgingival cups.

—*S. E.*

418—Vianden, J.: Behandlung paradentischer Kranheitsformen mit Pyoktanin. Treatment of periodontal disease with Pyoktanin, in: Schweizer. Monats. f. Zahnheil., 45: 38-43. January, 1935.

Pyoktanin is the name given to two aniline dyes. One is a blue aniline dye, methyl violet and the other is a yellow aniline dye, auramin. The blue dye, methyl violet is superior to the yellow in antiseptic action. Therefore, in cases where the blue dye is thought to be too radical in its action, the yellow is used.

It has been recommended in pyorrhea, stomatitis and in ulcerative conditions of the gums. Pyoktanin is indicated mainly in diseases which are a result of excessive accumulation of calculus and in neglected mastication. Pyoktanin solution is applied to the gums after the calculus has been removed to allow the Pyoktanin to get at the septic material and to subsequently promote the regeneration of the gum to the original condition of health.

To aid in subduing pain in extremely sensitive and painful cases, a prescription is given. It consists of a teaspoonful of Folia Salviae (leaves of Labiatae) brewed as a tea in a cup of water. Frequent rinsing until the pain subsides. Then the calculus is removed and the Pyoktanin is applied postoperatively.

—*N. K. B.*

419—Zemsky, J. L., D. D. S.: Important adjuncts in the treatment of periodontoclasia, in: D. Outlook, 22: 86-90, 392-395. 1935.

The treatment of periodontoclasia by heliotherapy occupies a significant place "for it offers an effective means of inhibiting the growth of microörganisms in the tissues. This is accompanied by a process of purification which results in healthy and resistant structures not conducive to the development of germs. The precise action, however, of ultraviolet rays on tissues are not yet fully understood.

The quartz lamp is satisfactory in treating pyorrhea. The lamp is applied to the surface of the gingival mucosa overlying the root, labially and lingually. No more than three or four teeth are irradiated at a time. Pressure on the tissues dehematizes them and the action of the rays is enhanced. A time schedule based on Eilstein and Lake is presented.

It should be remembered that heliotherapy is merely an adjunct to other measures. Prophylaxis is indispensable in all cases of pyorrhea no matter what therapeutic measures are undertaken and should always precede the particular treatment. However, after x-ray treatment the tissues have lost some of their physiological resistance and should not be immediately subjected to instrumentation.

X-rays have also been used with some success in treating pyorrhea. The author presents the technique of x-ray irradiation including dosage, distance and precautions. "Very satisfactory results of x-ray therapy in treating periodontal diseases are reported by various writers; according to the findings of some this agency would seem to be a method of choice, because with its use there is a possibility of obtaining clinical results with no injury either to the diseased structures or the overlying parts."

—*G. H.*

420—Zeses, M. H.: Diabetic patients and surgical intervention, in: Greek D. Rev. June, p. 21, 1935. Abst. in: J. Am. Dent. A. Sept., p. 1625. 1935.

Dr. Zeses contends that, in the treatment, surgically, of diabetic patients, the condition of the tissues needing attention should get first consideration rather than the diabetic condition. The opening of an abscess in such a patient would not injure the patient, but rather would benefit him by reducing the ills that the system has to

cope with. The chance of spreading the infection is strong, but with present-day knowledge of how to prepare the patient for the operation, the operation should be done.

—*Galonos*

(g) Home Care and Prophylaxis

(*Continued from 6(2): 103. July, 1935*)

60—Hirschfeld, I., D. D. S.: **The toothbrush—its use and abuse,** in: D. Items of Int. 57(1): 9-21, (5)383-391, (6)479-484, (8)663-674, (10)880-890, (11)969-974. 1935.

Dr. Hirschfeld presents a long series of cases with related pictures illustrating the various factors involved in the group of conditions known as cervical wasting.

Wasting, or wearing away of tooth structure, when found, tends to be most marked in those regions of the mouth where the abrasive action and motion of the toothbrush is most concentrated and the stroke most free.

Prior to the almost universal adoption of the toothbrush there were very few cases of wasting seen.

Wastings are found most frequently at the cemento-enamel junction, although they are by no means confined to this area, occurring anywhere on the crown or exposed portion of the root.

The phenomenon is divided into three types: (1) abrasion (due entirely to mechanical action); (2) Erosion (due to mechanical action superimposed upon a previous chemical action); (3) Caries. The last two are fostered by lack of tooth brushing, the first being due entirely to the over zealous use of the toothbrush.

The characteristics which distinguish the different conditions are: In abrasion, the floor is hard, highly polished and smooth. The outline is regular and wedge shaped with the deepest part at the cemento-enamel junction. In erosion, the outline is regular, the floor hard and gritty like ground glass. In caries, the outline is irregular, the floor is soft and discolored, and the gingivae inflamed.

The treatment involves correction and restoration. A correct diagnosis as to the etiology must be made. The aggravating conditions must be eliminated and corrected. This usually involves a change in tooth brushing methods. Often it is necessary to fill these cavities. The first step in prevention is to stop gum recession.

—*S. H. S.*

61—Parmly, L. P.: **Periodontia,** in: D. J. of Australia; Abst., in: D. Cosmos, 7: 343. May, 1935.

The author developed a consciousness of the fact that cleaning the teeth is a very important service to his patients. He felt that it helped to lengthen the period of usefulness for dentures.

He recounts old-practice system of leaving the cleaning to last twenty minutes of the appointment.

—*S. E.*

62—Pincus, Cecil: **The toothbrush,** in: Australian J. of D. 39(15): 153-155. 1935.

The main faults with the average toothbrush are: (1) Brush too large; (2) Brush too soft; (3) The same brush used night and morning; (4) Brush usually not pushed through interproximal spaces.

The author recommends a mode of brushing substantially the same as the Charter method.

—*S. H. S.*

(h) Vincent's Infection

(*Continued from 6(2): 109. July, 1935*)

202—Belding, P. H., D. D. S. and Belding, L. J., M. D.: **Why Vincent's infection?** in: J. Am. Dent. A. 22(12): 2114-2120. Dec., 1935.

The authors maintain that gingivitis, Vincent's infection, and periodontoclasia are all manifestations of the same disease, specific in nature and caused by a fuso-spirochetal complex. Gingivitis is a milder form of the so-called Vincent's infection, while pyorrhea is an advanced form or superimposed condition.

Eleven observations are noted concerning the ever-presence of the fuso-spirochetal complex in all cases of periodontal disease.

In proof of this theory the Drs. Belding

advance the fact that all these gingivitides respond to specific therapy. They recommend that "anyone interested in this problem should prove or disprove to his own satisfaction the specific nature of common gingivitides by treating a few cases with the following prescription:

Solution of potassium arsenite (Fowler's)	40 c. c.
Glycerine	60 c. c.
Neoarsphenamine	0.9 gm.

This is not a panacea and should be used as indicated in association with surgical and mechanical cleansing. The patient is instructed to wet his toothbrush in this solution and work it carefully into the subgingival and interproximal areas once a day."

—*S. H. S.*

203—Bulleid, Arthur, L. D. S.: **Vincent's infection**, in: D. J. Australia, 7: 71-75. 1935.

Vincent's infection or acute necrotic gingivitis is associated with stagnation in the mouth which suggests why, in the mouth, the disease begins in the lower third molar region. The symptoms are necrotic ulceration with pseudomembrane formation, gland enlargement, pyrexia, furred tongue, offensive breath and general malaise. The blood picture in the absence of secondary infection, shows a polymorphonuclear leucopenia with a relative lymphocytosis.

The author briefly discusses the bacteriology of the Vincent's organisms namely B. fusiformis and S. Vincenti. There is no evidence to support the contention that these two organisms represent two phases of the same organism. If aerobic cultures are made from the ulcerated patches an almost pure growth of strep. viridans is generally found. Treatment is both local and general. Locally the author advocates pure carbolic acid, 50% trichloracetic acid, chromic acid (5-10%), and prophylaxis after the acute phase has subsided. The general treatment consists of giving an iron and potassium chlorate mixture, and in the case of children, cod liver oil.

—*G. H.*

204—Beltrami, Prof. G.: **La stomatite de Vincent.** Vincent's stomatitis, in: Semaine Dentaire, 17: 60-65. 1935.

The etiology of Vincent's disease is dependent on two factors; bacterial infection and the resistance of the host, both local and general. Even in a healthy mouth a local irritation will cause a banal lesion and an inflammatory state. The ever present fuso-spirillary anaerobes find such a medium a good culture field and will then start to proliferate. Vincent's infection is never primary in nature, always secondary. If this is so, however, why the occurrence of epidemics of trench mouth during the war, in hospitals, institutions, etc.? Such epidemics occur because the general systemic condition plays a prime role in Vincent's. Groups exposed to the same debilitating conditions of climate, poor food, hard work, fatigue, etc., are prone to Vincent's. That is what happened in the war. Troops living on proteins, carbohydrates, and the hard life of the trenches succumbed to the infection. Troops well nourished with fresh vegetables, etc., had a low incidence of Vincent's. Dietary regulation and general systemic rehabilitation should, therefore, accompany local curettage and medication with oxidizing agents as the treatment for Vincent's disease.

—*G. H.*

205—Bettman, M., and Ratte, G.: **Vincent's infection.** L' infection de Vincent, in: J. Canadian D. Ass'n., 1: 283-285. 1935.

The author presents a brief resumé of the clinical symptoms of Vincent's infection. The destructive type is characterized by ulceration and necrosis of the marginal gingiva. The hypertrophic type is marked by subjective symptoms such as fever, malaise, etc., superimposed upon the same clinical symptoms as the destructive type, except for hypertrophy of the gums with or without gangrene. Bacteriology and treatment are also presented.

Vincent's will not occur in a mouth where the tissues are healthy and free from irritation. The Vincent's organisms introduced into such a mouth will probably not produce the disease.

—*G. H.*

206—Clough, P. W., M. D.: **The treatment of fuso-spirochetal infections**, in: Med. Clin. of N. A., 18: 1711-1724. May, 1935.

The author deals with the fuso-spirochetal infections in general, and more specifically with Vincent's angina and Vincent's stomatitis. Although rare, the organism may invade any part of the body causing abscess formation and gangrene.

The classical symptoms of Vincent's stomatitis are presented together with the morphological, staining and pathogenic characteristics of the organisms. Microscopically the ulcer of Vincent's infection is made up of three fairly distinct layers. 1. "A superficial necrotic layer consisting of cellular detritus, masses of mouth bacteria, and varying numbers of spirochetes and fusiform bacilli." 2. "An intermediate layer of acutely inflamed tissue in which the fusiform bacilli predominate." 3. "A deep layer containing masses of spirochetes, which appear to be the actively invasive agents." There is little or no leucocytic infiltration of the tissues.

Treatment involves 1) the establishment of mouth hygiene, 2) gentle local cleansing methods, 3) applications of oxidizing agents, of which sodium perborate is probably the best. This is followed by the application of arsphenamine as a paste, or acid potassium bismuth tartrate in 10% solution or 30% ointment, 4) In severe cases neo-arsphenamine injected intravenously is used efficaciously.

It is noted that there is no single treatment that is successful in all cases.

—S. H. S.

207—Dobbs, E. C., D. D. S.: **Chromic acid,** in: D. Cosmos, 77(1): 92-93. 1935.

The author claims that the rationale of using chromic acid in Vincent's infection cannot be substantiated.

He states:

"Chromic acid does not liberate free oxygen gas in the oral cavity.

"The acid action of chromic acid can decalcify the hard tissues of the teeth.

"This decalcification may produce sensitiveness about the necks of the teeth."

This decalcification may predispose the teeth to dental caries.

"The teeth should be protected from the corrosive action of chromic acid. This may be accomplished by covering the teeth, in the area to be treated, with cavity varnish."

—S. H. S.

208—Downes, J. R., M. D.: **Trench mouth,** in: Delaware State Med. J. 7: 180-181. 1935.

The author calls to attention a widespread increase in the prevalence of Vincent's infection. Use of common drinking cups and poorly cleaned eating and drinking vessels play a large part in the spread of the disease. Public health campaigns impressing this fact upon the community will bring about public coöperation on this score and aid in decreasing the incidence of trench mouth.

—G. H.

209—Downs, W. G.: **Allergy and the oral cavity,** in: D. Outlook, 23: 127. August, 1936.

The author suggests that since foci of infection elsewhere may through allergic phenomena affect the mouth, dentists may, as a result, be of real aid to the internist.

Many mouth pathologies have an allergic basis. Specific diseases like gonorrhea, often affect the organism in such a way that allergic skin reactions take place. In cases referred to by the author there seems to exist a correlation between gonorrhea and Vincent's infection. Avitaminoses and endocrine disturbances may do the same. Generally, trench-mouth is due to lowered tissue vitality and lowered oxygen tension, so that organisms normally harmless become virulent. The allergic response in the form of Vincent's infection is then only one of several causes for this condition.

The need for complete case records and clinical laboratory tests will bring complete understanding of diseases and the delivery of a broader health service.

210—Fleming, W. C.: **Diagnosis of Vincent's infection,** in: D. J. Australia, 7: 179-181. 1935. Austral. D. J. 38: 6-12. 1935. Selected article from Jour. Southern Calif. Dental Ass'n.

The mere presence of the fuso-spirochetal organisms in the lesions does not necessarily mean that the organisms are the cause of the disease. No work has ever proved that the fuso-spirochetal symbiosis is the main etiologic factor in Vincent's. There has been some evidence that these organisms are sec-

ondary invaders. Moreover, they are present in non-diseased mouths. The diagnosis of Vincent's infection rests not upon any positive laboratory report concerning the presence of spirochetes but on the clinical signs, i. e., ulcers and pseudomembrane, pain, bleeding, fever, malaise, etc. Cases not responding to treatment should be referred to a medical man as symptomatic of an abnormal systemic condition.

—*G. H.*

211—Levin, M.: **Vincent's organisms in rural school children,** in: J. Michigan State D. S. 17: 706. 1935.

Smears were made from the mouths of 166 children in two rural schools of Ottawa County, Michigan. Stained with gentian violet the smears showed 91.5% of the total with Vincent's organisms in such numbers as would justify a diagnosis of Vincent's infection. Of 14 children whose smears were negative 50% had dental caries, 6 had fillings and 1 had no caries. Of the total number 82% had caries and 19% gingivitis.

—*G. H.*

212—Levington, Herbert, D. D. S.: **Review of views on Vincent's infection,** in: International J. Orthodontia, 21: 1171-1176. 1935.

The status of Vincent's infection as evaluated by the medical and dental professions is still doubtful. Four articles in the 1933 J. Am. Dent. A. are compared with the description of the disease in Mead's "Diseases of the Mouth." The articles are those of Smith, Merrifeld, Rosenthal, and Becks. The points in dispute are the etiology, bacteriology, location, and treatment of the infection. Becks doubts the validity of Tunnicliff's anaerobic cultures of the fusiform-spirochetal symbiosis. Tunnicliff's experiments have had little or no corroboration by other workers. Bacteriologically an accurate classification and description of the types of the fusiform bacilli and spirochetes present in the mouth is not to be had. As for treatment the above authors list no less than twenty-two different chemicals in the treatment and are of varied opinions as to operative procedures.

A diversity of opinions exist among medical writers on the subject. Lichtenberg, Wiener, and Luieck report their inability to cultivate Vincent's organisms and demonstrate their pathogenicity. Workers in the field present such a diversity of opinions on etiology, bacteriology, and treatment of Vincent's infection that it seems obvious that the status of the disease is still an open question.

—*G. H.*

213—Lyons, D. C., and Coffelt, O. T.: **Preliminary evaluation of a solution of cerous-ceric-chromate in treatment of Vincent's infection and other oral diseases,** in: D. Digest, 41: 201-202. 1935.

The authors are of the opinion that the use of spirocheticides in the treatment of Vincent's infection is not desirable because of the difficulty of administration and the fact that arsphenamine treatment often results in Vincent's angina.

Of the oxidizing agents chromic acid is the most powerful but is objectionable because of its caustic powers. The unstable nature of the compound also limits its use. Cerous-ceric chromate is a chemical combination of ceric oxides with chromic acid at high temperatures. It is a stable solution and a strong oxidant. The authors report it an excellent means of combating chronic and acute Vincent's infection. It does not stain, is not disagreeable in taste and has no odor. Further tests are advised.

—*G. H.*

214—McLean, D. W., **Vincents infection, or trench mouth,** in: Hygeia, 13: 540-541. 1935.

This is a brief account of Vincent's infection intended primarily for the layman. It involves a short description of the organisms associated with the condition, the clinical picture, and the treatment.

—*S. H. S.*

215—Palmer, B. B., and Carr, M. W.: **Medico-dental case records; fusospirochetal angina.** in: J. D. Research, 15: 93-99. 1935.

The author's cite a case of primary chronic periodontal fusospirochetal infection which was directly extended into a fatal secondary lesion. The patient, a white female, 55 years of age, had been suffering

from recurrent attacks of Vincent's stomatitis for six months. Despite local treatment the infection became chronic. Elimination of the lower right third molar which was believed to be the focal point, resulted in exacerbation. A gangrenous stomatitis and a facial and submaxillary cellulitis resulted. After an unsuccessful drainage operation death resulted. The authors conclude that "Death from fusospirochetal angina may be due to extension of the local pathologic process involving the glottis (edema), to toxemia or hemorrhage arising from extensive sloughing areas, or to the occurrence of metastatic lesions elsewhere in the body."

—*G. H.*

216—Rosebury, T., D. D. S., and Siegel, E. H.: **Notes on current research. Recent studies of Vincent's infection and related diseases,** in: J. N. Y. Acad. Dentistry, 2(1): 27-32. 1935.

The latest experimental work of Smith, Varney, Proske, and others indicates that lesions comparable to those of fusospirochetal disease in man can be produced only with particular combinations of four species of organisms; T. microdentium, B. fusiformis, vibrio viridans, and non-hemolytic anaerobic streptococcus.

Several new contributions have been made towards a clearer understanding of the factors which predispose to this mixed infection. In 1933 Wallace and Robertson produced Vincent's angina in dogs by the injection of the drug scillaren B. This drug produces a leukopenia and local dilatation of skin vessels. Hanke's work and that of Kirkpatrick and Clemens indicates that lack of Vitamin B may play an important role as a predisposing factor to Vincent's infection.

—*G. H.*

217—Schamberg, M. I., D. D. S., M. D.: **A new therapeutic preparation in the treatment of fusospirochetal infections,** in: D. Items of Interest, 57(4): 297-299. 1935.

The author briefly goes over the etiology and symptomatology of Vincent's infection. He claims that simple oxidizing agents are the treatment of choice.

218—Wisan, J. M., D. D. S.: **Public health aspects of Vincent's infection,** in: J. Am. Dent. A. 22(II): 1765-1776. Oct., 1935.

Vincent's infection being a "specific infection caused by definite and well-known types of micro-organisms," demands the united attention of health officials, dentists and physicians. "Government reports, newspaper items, medical journals and correspondence with physicians give proof that epidemics are occurring throughout the country and that the disease is on the increase in many communities."

The article is most comprehensive and covers carefully etiology, diagnosis, treatment, personal care, and public health policies. Under the latter is considered the duties of school administrators, dentists, and physicians regarding Vincent's infection.

219—Wood, T. B., M. D.: **Soap as a therapeutic agent in Vincent's,** in: Med. Times and Long Island Med. Jour., 63: 608-612. 1935.

The author recommends soap as a mouth wash, 1:250 dilution, in the treatment of Vincent's infection of the gums and throat. He finds that soap is strongly antiseptic against pneumococcus, streptococcus, meningococcus, gonococcus, B. diphtheria, influenza B., and the spirofusiform bacillus of Vincent. It is also a detoxifying agent against the virus of diphtheria, tetanus, leprosy, scarlet fever and poliomyelitis.

Soap is dependent for its action on its saponifying ability, its ability to lower surface tension, dialyzing ability, detoxifying property and diffusing ability. The antiseptic quality of soap varies according to the temperature of the solution, fatty acid radical, fatty acid saturation, absence of foreign substances and the degree of dilution. The local action on mucous membranes did not show any deleterious effects. Five case reports of Vincent's infections of mouth and throat successfully treated by soap mouth washes are presented. Stearates and oleates have the greatest germicidal efficency.

—*G. H.*

(i) Miscellaneous

(Continued from 6(2): 112. July, 1935)

133—Albright, H. L.: **Carcinoma of the mouth with special reference to treatment,** in: J. of Radiology, July, 1935. Abst. (condensed) from J. A. M. A. 105(11): 916. Sept. 14, 1935.

More than 90% of malignant oral tumors are epidermoid carcinomas, of the adult resistant type. Of those of the buccal epithelium, only the basement cell variety has power of reproduction and invasion. Other cell changes represent stages of degeneration. Epulis is semi-malignant. Leukoplakia is the most important oral precancerous lesion. "All cases of leukoplakia are curable." Enlarged cervical nodes should be considered cancerous and once the nodes have been invaded the chances of a cure are practically gone. Early diagnosis is more important than any other single factor and best results in treatment are held by coöperation between surgeon and radiologist.

134—Bland and First: **The relationship of the stomatological clinic to the maternity dispensary,** in: Med. Rec. 281-284. Mar. 1935.

The author emphasizes the importance of the relationship of stomatology to obstetrics under the following three heads;

1) The influence of pregnancy on diseases of the mouth.
2) Influence of diseases of the mouth on pregnancy.
3) The influence of diseases of the mouth on puerperium.

Among the many influences of pregnancy on the mouth is that on the gingivae. Beginning about the fourth month of pregnancy and continuing until parturition there is a swelling and hyperaemia of the gingivae. This may be mild and cause no discomfort, or it may be severe and cause them to become oedematous, spongy and hemorrhagic upon the slightest touch. In some cases the teeth become loose and fall out; in others there is an hypertrophy accompanied by the appearance of polypoid excresences between the incisors, known as pregnancy tumors.

—*S. H. S.*

135—Bresler, H. E., D. D. S.: **Dissertation on gingivitis gravidarum,** in: D. Items of Interest, 57(7): 605-613. 1935.

Dr. Bresler presents the different manifestations of pregnancy gingivitis including the occurrence of tumors on the gingivae which disappear after parturition. He also outlines the various observations and opinions of other workers on the same subject.

—*S. H. S.*

136—Camille, André: **Soap considered as a gingival poison,** in: Rev. Odontolog. April, 1935. Abst. in: J. Am. Dent. A.: 1262. 1935.

The use of soap dentifrices has dominated dental hygiene in the present generation. The author gives a historical survey of the early literature on this subject. He states that soap has a caustic and irritating action when in contact with the oral mucosa. He compares the tissues of the skin and the mucosa in connection with their relative reactions to soap, pointing out that the mucous membrane has no epidermal layer to protect it from the action of the soap. The epithelial lining of the mucous membrane has no resistance against soap. The author explains the mechanism of soap's harmful action on the muciparous glands. It is a slow chronic poison concentrating its action on the dental interstices. Daily use of soap dentifrices is responsible for chronic lesions and lowered tissue resistance. The mouth is normally acid, but constant use of soap changes the dental interstices to an alkaline state. Permanent alkalinization produces simple inflammatory lesions and a good medium for bacterial infections. Experiments were conducted to test the action of soap on dental cement. Cement was injured by twenty-four hours' contact with the soapy solution. Certain deductions are made concerning the use of soap dentifrices and further experiments on this subject are believed to be necessary.

137—Crowley, Mary C. and U. G. Rickert: **Method of estimating bacterial content of mouth by direct count,** in: J. of Bacteriology, Oct., 1935. Abst., (condensed) from J. A. M. A., 105(25): 2105. Dec. 21, 1935.

Crowley & Rickert used an atomizer to break up the larger clumps of bacteria, and a very dilute sodium hydroxide solution to

dissolve the mucin which held them together. Their method, which in other respects was similar to that used by Breed and Brew for making direct bacterial counts of milk, gave a better distribution of bacteria in smear and made more accurate count possible.

138—Downing, J. G. and J. B. Hazard: **Cutaneous moniliasis associated with oral thrush,** in: Archives of Dermatology and Syphilology. May, 1935. Abst., (condensed), from J. A. M. A. 105(3): 228. July 20, 1935.

An unusual case is described. Organisms from mouth and face, which showed markedly hyperkeratitic lesions, were yeast-like in structure, fermented certain sugars with the formation of acid and gas, produced segmented mycelia, did not form ascospores, did not coagulate milk and were pathogenic to rabbits. This type of Monilia is not usually present on normal skin.

139—Editorial: **Effects of Vitamin D.,** in: J. A. M. A., 105(13): 1042. Sept. 28, 1935.

Commenting on an experiment of E. W. Fish, who fed dogs excessive amounts of calcium and vitamin D to determine whether the dentin of the animals was affected thereby, the Editor (J. A. M. A.) says that although chemical analysis showed no significant increase of calcium in dogs' teeth after seventy days, the case might have been different in humans, since there is normally less calcium in teeth of dog than of man. It is of interest to periodontists that while dentin showed no change, X-ray showed marked increase in opacity of bones.

140—Foreign letter: **Malignant tumors of mouth,** in: J. A. M. A., 105(9): 732. Aug. 31, 1935.

Professor Lindemann, director of West German Clinic in Dusseldorf studied more than two hundred cases of oral carcinomas and sarcomas. Lindemann found that irradiation is useful to reduce growth to an operable size. Schreus considers irradiation preferred treatment in carcinoma of upper jaw and in lingual and pharyngeal tumors. He has an increasing preference for radium treatment within the tumor where there are glandular metastases.

141—Haggard, Howard W., M. D. and Greenberg, Leon A., Ph. D.: **Breath odors from alliaceous substances, cause and remedy,** in: J. A. M. A. 104(24): 2160. June 15, 1935.

Authors recognize that breath odors may arise from pathologic conditions in structures of mouth and respiratory tract although pathology is not always present. Cause of odor may be systemic but authors' evidence indicates that the source is generally local in mouth or respiratory tract. Determination of source is essential to effective therapy and an experimental investigation was made into the source of odors arising after eating onions or garlic. Author's conclusions follow: "The odor given to the breath by onion or garlic comes from the essential oil contained in these vegetables. The oil does not, as has been suggested, reach the breath from aeration of the blood in the lungs, from pulmonary secretion, from salivary secretion, or in air passed from the stomach. It arises solely from particles of onion or garlic retained in the structures about the mouth.

Quantitative experiments demonstrate this fact.

The particles from which the odor arises cannot be removed completely by mechanical means; brushing the teeth and tongue and washing the mouth with soap and water fail to deodorize the breath. Similarly, washing the mouth with a 30% solution of alcohol is ineffective.

The breath can be immediately and completely rid of the odor by washing the teeth and tongue and rinsing the mouth with a solution of chloramine. The chlorine liberated in the mouth reacts chemically with the essential oils and deodorizes them. It is probable that many cases of foul breath from other causes would be amenable to to the same treatment."

142—Hewat, Robert E. T.: **Preventive dentistry and the dentist,** in: New Zealand D. J., 31: 344. November, 1935.

In this presentation, the author feels that dentists talk preventive dentistry but never correlate theory with practice. With this idea in mind, he supplies definite information and procedures in prophylaxis, periodontoclasia, oral examination, fillings and orthodontia which are preventive in pur-

pose and character. The author concludes with the idea that complete prevention is still impossible, but worthwhile efforts can be made to give the best possible service under the situation as it exists.

143—Jaccard, René: **L'orientation des idées odontologiques et la paradentose.** Orientation of odontological ideas and paradentosis, in: Semaine Dentaire. 17: 358-364. 1935.

Dr. Jaccard in his opening address to the third session of the International A. R. P. A. discusses the trend to regard stomatology as a medical specialty. It is evident that while treatment of oral diseases requires the same basic knowledge as other medical specialties one can not divorce stomatology from dentistry and its technical sides. Odontology, or dentistry, must be considered as requiring the basic medical sciences in addition to a highly technical training and manual dexterity and as such is part of medicine, yet is entirely outside of it.

It is unimportant whether one adopts the terms stomatology, dentistry, oral health service, odontology or another designation. It is more important to create a close coöperation between physician, medical specialists and dentists. In the study of paradentosis one is brought face to face with the importance of the systemic relationships of the oral cavity. Periodontology should be included in dental education by the side of orthodontia and prosthesis.

The common goal that we are striving for is the spirit of a medical synthesis and the progress of medicine and dentistry and the sciences upon which they are based.

—*G. H.*

144—Mayo, C. H., M. D.: **The unity of preventive dentistry and the newer preventive medicine,** in: Military Surgeon, 76: 289-293. 1935.

The author feels that dentistry is a specialty of medicine and that the practice of preventive dentistry is the practice of preventive medicine. Dentists should know much about medicine as a whole and all physicians, conversely should know more about dentistry. It is only through medical and dental coöperation that the problem of focal infection may be completely investigated.

A plea is made for the dissemination of knowledge concerning preventive dentistry and medicine to the public. The medical and dental corps of the army and navy through instruction to its personnel may play a vital part in the dissemination of such knowledge.

—*G. H.*

145—Merritt, A. H., D. D. S.: **A program for postgraduate instruction in periodontia,** in: J. Am. Dent. A. 22: 785. May, 1935.

The author feels that in order for the general practitioner, now in practice for some time, to measure up to his responsibilities, a provision for postgraduate work in periodontia must be made. The responsibilities are: 1) to make a differential diagnosis of all the more common periodontal diseases, 2) to treat these diseases in their earlier manifestations, and 3) to take such steps as may be necessary to prevent their development.

The courses are to be done under the auspices of the dental profession and should have as their goal "to give the practitioner a working knowledge of periodontia."

There follow three arrangements whereby such a course could be given. They include: 1) a school-supervised course—with regular sessions, 2) society-sponsored course —with demonstration-lectures, 3) local society-sponsored—with clinics held at the instructor's *office*.

A full outline for a periodontia course is presented.

146—Merritt, A. H., D. D. S.: **Preventive dentistry, its possibilities and limitations,** in: J. of Canadian D. A. p. 14. January, 1935.

The highest achievement of the healing art is its ability to prevent disease. Diseases are of two types, a) those cured without leaving any evidence, b) those leaving irreparable changes after cure. Preventive dentistry is important, for most dental conditions leave irreparable conditions that are only temporarily patched up. Preventive dentistry consists of all measures employed to protect the teeth and supporting structures from disease. These measures are local and systemic. In the preventive scheme the systemic must and should precede local

measures. Local measures control disease spread.

Local measures are:

1) periodic examinations—aid foresight and prevention of disease development,
2) normal occlusion establishment (orthodontia)—makes possible normal function and resultant tissue health,
3) mouth hygiene—supposedly staves off disease—never shown experimentally,
4) prophylactic odontotomy — controls tooth decay, but does not prevent it,
5) normal tooth form—poor contacts and poorly reconstructed anatomy predisposes to caries and periodontal disturbances,
6) gum stimulation—through function (rare today) or through artificial stimulation.

Systemic measures prevent disease onset.

Maintenance of correct acid-base balance through correct diet prevents dental disease by 1) decrease in the base content, with slight acidosis resulting, increases liability of enamel to decay, 2) increase of base content to alkalosis increases liability to periodontal disturbances.

Diet disturbances:

1) fetus insufficiency of diet due to maternal insufficiency still experimentally unproven,
2) lactation period. Maternal organism protects itself at the expense of the offspring,
3) diet: adequate calcium, phosphorus, calcifying vitamins and radiant energy are essential factors in good tooth structure,
4) hormonodystrophy may alter the absorptive process,
5) activity and sunlight alter the acid-base balance by raising the acidity. Children show high acid—high caries relationship.
6) ill-health—alters the nutritive process to the detriment of dental health.

Regulation of the diet, therefore, takes on prime importance. It should be done through the active collaboration of the dentist with the physician.

—S. E.

147—Morrison, D. D. S.: Protean pyorrhea, in: Dent. Mag. and Oral Topics, (11): 1095-1096. 1935.

The author lists 110 synonyms for pyorrhea compiled in the course of his readings on the subject.

—S. H. S.

148—Pyorrhea: A lecture, in: J. Am. Dent. A., 22: 695-697. 1935.

This is a lecture accompanied by pictures showing the progress of pyorrhea from its inception. It is designed to educate the layman. The lecture with colored slides, can be purchased from the Bureau of Public Relations, of the American Dental Association.

—S. H. S.

149—Query: Re. Black tongue and halitosis, in: J. A. M. A. 105(4): 303. July 27, 1935.

Man of forty has for many years had thick grayish coating on tongue with foul odor. Saliva is slightly acid. Patient has vaso-motor rhinitis. Alkaline washes, boric acid, internal administration of iodine have been tried without noticeable benefit and advice is asked as to treatment and prognosis. Answer states that bad odor is probably due to growth of fungi at base of elongated papillae or beneath the tongue coating. This may be a case of black tongue. Bad odor is usual in some forms of rhinitis but not in this particular type. Repeated and thorough swabbing with hydrogen peroxide followed by warm physiologic sodium chloride solution usually helps, together with persistent use of soft brush. But this latter is difficult because of location of deposits at base of tongue. For obstinate black tongue, Prinz ("Diseases of Mouth and their Treatment") advises swabbing with ten to fifteen per cent salicylic acid in alcohol and glycerin equal parts, this followed by normal saline irrigations. Scrapers may be used but their use is likely to be followed by recurrence.

150—Query: Re. Black discoloration of the incisors, in: J. A. M. A. 104(2): 138. Jan. 12, 1935.

Blackening of incisor teeth of child of three and a half may be easily removed if defect is a stain, otherwise dark teeth are

probably pulpless. Mottled enamel would not be found in temporary teeth and diet in the present case being essentially correct except for milk deficiency, condition is not likely to be due to this cause. In the absence of further etiologic information cleaning and polishing is recommended by the editor.

151—Query: Re. Burning pain and blood circulation in the mouth, in: J. A. M. A., 105(20): 1629. Nov. 16, 1935.

Answering query regarding burning pain in mouth and throat of two patients wearing false teeth of gold and vulcanite respectively, in both of whom, symptoms are relieved by removing plates, editor says that almost invariably the sensation is due to impaired blood supply from pressure of denture over anterior and posterior palatine foramina. Relief will be had if impressions for plates are prepared so as to relieve this pressure, before proceeding with making dentures.

152—Query: Re. Chronic glossitis, in: J. A. M. A., 104(4): 339. Jan. 26, 1935.

Information is asked as to etiology and treatment for patient with sore tongue and red tender mucous membrane. Tongue is not coated. Patient formerly had secondary anaemia which improved under treatment.

Answer—Symptoms are suggestive of a chronic disease known as Moeller's Glossitis (see article by F. G. Harris, Jour. of Cut. Dis., 33: 742, 1915). Etiology is usually obscure. In Moeller's cases patients had intestinal worms but other writers report no such infestation. Engmann and Weiss have reported a case in which endamebas were found in tooth abscesses and which was cured by extraction of teeth and treatment with emetine. Another authority attributes all cases to pernicious anaemia and advises watching blood carefully and continuously. All foods should be soft and if necessary tongue may be anaesthetized before eating. Sharp corners of teeth should be smoothed off. Judicious use of x-ray and ultra-violet radiation may give temporary relief. Tomato juice and yeast by mouth and liver extract intramuscularly might be beneficial. Vitamin B has not proved helpful.

153—Query: Re. Diagnosis of syphilis, in: J. A. M. A., 105(13): 1062. Sept. 28, 1935.

Editor, commenting on diagnosis of syphilis based on meager symptomatic findings states that many oral lesions simulate syphilis. "The diagnosis of Vincent's infection can be easily corroborated by a smear."

154—Query: Re. Fissure of lip. J. A. M. A., 104(9): 770, March 2, 1935.

Correspondent asks for best method of curing central fissure of lower lip which has become steadily worse in spite of treatment "in every conceivable manner" over a period of years. Biopsy shows no malignancy. It is advised to make excision including a V extending down into lower lip some distance below red margin. This will permit a good plastic closure with no deformity if red margins are carefully approximated and sutured with fine silk.

155—Query: Re. Scrotal tongue, in: J. A. M. A. 104(5): 419. Feb. 2, 1935.

Furrowed or scrotal tongue (lingua plicata) is usually congenital and often familial and in its milder forms is fairly common. Condition is permanent and only significant when food particles, etc., collect in crevices, which may predispose to epithelioma. Querist seems to suspect connection between scrotal tongue and hypothyroidism but there is no recognized direct connection between the two.

156—Query: Re. Sinus in gums from toothpick trauma, in: J. A. M. A., 105(8): 618. Aug. 24, 1935.

Man of thiry-seven, two years ago ran a toothpick into the mucous membrane just posterior to the anterior teeth. He thought he had removed all of the toothpick but area swells from time to time and drains. Sinus is now present extending about ¼ inch into the bone. All other symptoms are essentially negative, even X-ray showing no gross bone change.

Editor suggests that the splinter may be present without showing in X-ray. Facts should be ascertained by raising a mucoperiosteal flap exposing territory involved, which can then be explored and irritant removed.

157—Query: **Re. Stains on teeth.** J. A. M. A. 105(2): 141. July 13, 1935.

Replying to question regarding dark stains on children's teeth, editor states that such green and black stains are often growths of fungi and most frequently associated with ropy saliva. Plenty of water, milk and citrus fruits may reduce the amount of stain, and frequent brushing is most desirable.

158—Query: **Re. Ulcerations in mouth during menstruation,** in: J. A. M. A. 105(11): 905. Sept. 14, 1935.

Woman of thirty-five has for many years suffered constantly from ulcers on tongue and buccal mucosa. They are two to five mm. in diameter, round and slightly depressed and covered with grayish membrane, slightly resembling thrush. Removal of membrane leaves raw bleeding surface. Smears show pus cells but no definite organisms. As a rule condition is aggravated just before menstruation, and mother was similarly affected up to forty years of age. Patient suffers from constipation, formerly had malaria; blood examination reveals four million red cells, 80% haemoglobin, no white count made; urinalysis negative, and basal metabolism and gastric analysis have not been made. Many doctors have diagnosed and treated case variously over seven-year period, hyperacidity, intestinal intoxication, nervousness, endocrine dysfunction and incorrect diet have been suspected and treated without benefit. Editor thinks small ulcerations at period described suggest possibility of chronic relapsing agranulocytic angina, in which case there would be a periodic sharp drop in polymorphonuclears. Advises frequent white cell count and refers querist to articles of Henry J. Jackson and Dudley Merrill, and of W. P. Thompson on the subject of Agranulocytic angina associated with the menstrual cycle, in New England Journal of Medicine 210: 175-6, Jan., 1934. If a periodic leukopenia is determined, pent-nucleatide and gonadotropic factor of pregnancy urine may prove beneficial. (Jackson and Merrill above.) Basal metabolism should be determined and if low or even normal, dessicated thyroid in small daily doses may be helpful.

159—Simpson, F. E.: **Radium dosage in carcinoma of the tongue,** in: American Jour. of Roentg. and Rad. Therapy, July, 1935, p. 63. Condensed from Abst. in: J. A. M. A., 105(13): 1071. Sept. 28, 1935.

Simpson emphasizes the importance of hygienic condition of mouth and teeth as part of chain of details essential to success in treatment of carcinoma of the tongue.

160—Stern, M. M., D. D. S.: **Prevention of periodontal disease by the general practitioner of dentistry,** in: D. Items of Interest, 57(6): 485-488. 1935.

By instituting a thorough prophylaxis at regular and frequent intervals the general practitioner can help prevent periodontal diseases, Periodontoclasia is treated by simple measures. A summary of the histories of 36 patients treated over a period of ten years or more, shows that only thirty-three teeth were lost as a result of periodontal disease.

161—Tishler, B., D. M. D.: **Diagnosis and treatment of some referred periodontal cases,** in: D. Cosmos(6): 600-605, 1935.

Dr. Tishler cites six interesting cases that were referred to him by general practitioners. For the most part previous diagnoses were missed because simple dental causes had been overlooked for more complex factors.

In the first case, the large ingestion of yeast was the cause of gingival disturbances. Discontinuance of yeast cleared up the condition.

Case 2—Patient complained of very sore gums. It was found to be due to the rubbing in of plain salt.

Case 3—The proper use of the toothbrush and cutting down on smoking cleared up a gingival condition.

Case 4—Gingival massage helped a condition of inflamed and sore gingivae.

Case 5—Inflamed and bleeding gums cleared up by toothbrush massage and periodontal treatment of pockets.

Case 6—Patient in debilitated condition due to self starvation. Mouth lesions showed inflamed gingivae, alternate bands of

light and pink and dark red stripes across the tongue. Diagnosis of scurvy was made. The patient was prescribed a diet. He did not follow instructions and died shortly afterward of starvation.

—S. H. S.

162—Union Catalogue of Medical Literature, in: J. Am. Dent. A. 1935.

Two years ago the Institute of Medicine of Chicago made available an initial fund for the establishment of a union catalogue of medical literature in Chicago. The work of organization of this project was entrusted to a Committee on Coördination of Medical Libraries in Chicago, which had been appointed a year before. The first aim of the committee was to bring about closer integration of effort among the major medico-biological libraries, including dental and pharmacological, of the city (John Crerar Library, Universities of Chicago, Illinois, Northwestern and Loyola, Rush Medical College) to obviate unnecessary duplication, triplication or even quadruplication of special literature, and divert the economies achieved to a fuller representation of the world's medical literature in Chicago. The second aim was the creation of a union card index centrally placed, so that investigators in medical problems are able to locate the desired reference volumes with the least expenditure of time and energy. It is hoped that eventually the service of the catalogue will be made even more effective by the attachment of a permanent librarian and the availability of information by telephone. The task of achieving such a union catalogue and its cost in effort and money are much greater than the original estimates indicated. The work towards its completion is still in progress, but is pushed as quickly as possible. Every month these different medical libraries are sending in the cards of their new acquisitions, so that the catalogue is brought to date. It is hoped that in time the important holdings in the smaller medical libraries in the city will be represented in the catalogue. The final editing of this union index may not be accomplished before another two or three years have passed. Despite its present gaps and imperfections, it has already given valuable service to the medical librarians of the city, and to investigators who were aware of its existence. The union catalogue is now open to a wider medicinal public, which is invited to make use of it for reference and to coöperate in its further development and extension of service. It is to be found on the twelfth floor, or floor of the medical reading rooms, in the John Crerar Library. Further information concerning it may be obtained from the medical librarians indicated above, or from the central office of the Institute of Medicine of Chicago, 629 South Wood Street. Criticisms or suggestions should be sent to the latter. The chairman of the committee is Professor Otto F. Kampmeier, department of anatomy, College of Medicine, University of Illinois, Chicago.—*Science*, 82:54 (July 19), 1935.

163—Waite, C. H., B. A. and Gorrie, Rachael, M. S., Ala. State Board of Health: Gongylonema infestation in man, in: J. A. M. A., 105(1): 23-24, July 6, 1935.

Waite and Gorrey report rare case of man who had suffered for three months with sore mouth. He had been treated for Vincent's for several weeks when he appeared at laboratory with a worm which he had withdrawn with his fingers from the roof of his mouth. Antiseptic treatment had probably stimulated the worm to work out from its tunnel to surface of palate. Healing of opening was prompt and also that of tunnels through which worm had worked beneath mucous surface. Only general symptom experienced was nervousness which had completely disappeared as had all other symptoms six weeks after above incident. Worm was a male Gongylonema—grayish white, thirty-two millimeters long and seven mm. wide. From all the evidence infestation was thought to be accidental.

164—Weeks, H., D. D. S.: Report of a case of pemphigus foleaceus, in: J. of Perio, 6(1): 46-47. Jan., 1935.

The author presents a case of a woman aged 33 referred to her by a dermatologist.

Physical examination revealed a number of fillings and a bridge of two different metals on the lower right side. The tongue on the right side was swollen and had large yellowish-grey bullae filled with a yellow fluid. The left cheek opposite a

large copper amalgam filling was similarly swollen. Pain was associated with the condition.

It was thought the lesions were due to an electro-galvanic current set up by the different metals. All fillings and the bridge were removed, but the lesions persisted. The diagnosis was then made of Pemphigus.

The patient had complained of sore mouth to her physician some time previous. On the basis of a smear alone he had treated the case as Vincent's infection with arsenicals.

Dr. Weeks stresses the point that a smear alone is not enough for a diagnosis of Vincent's infection.

—*S. H. S.*

165—Withycombe, M. R.: Are the upper or lower anterior teeth first lost in pyorrhea?, in: D. J. Australia, 7: 289-291. May, 1935.

The author claims that the upper anterior teeth are lost before the lower anteriors in pyorrhea. The reason for this lies in the occlusion. In pyorrhetics the lower teeth are forced into two movements; a direct or orthal bite, or an anterio-posterior one. The latter is predominating and it is under the continual thrust of the lowers forward and upward that the upper teeth are dislodged.

—*G. H.*

166—Ziserman, A. J., M. D. Ulcerative vulvitis and stomatitis of endocrine origin, in: J. A. M. A., 104(10): 826. March 9, 1935.

Ziserman reports case of woman of twenty-three with periodically recurring ulcers of the mouth and occasionally of the vulva, the time and the duration of the lesions being definitely associated with the menstrual periods which were irregular; previous medical history otherwise irrelevant. The small ulcers were covered with grayish pellicle which left a raw bleeding state when removed. The usual situations were mucosa of lower lip, buccal surfaces, pharyngeal wall and the sides of tongue. The mouth lesions caused an aching to stab-like, pain.

Opinions vary in regard to etiology of these conditions and case is reported both because of rarity of condition and because therapeutic results obtained point to a possible endocrine etiology. Wasserman tests were negative but treatment was carried out, without any remission of symptoms. The only cessation was during the months of a pregnancy, lesions recommencing immediately after termination of same. Endocrine disfunction was naturally suspected as the etiologic factor. It was noted that lesions were present at the stage when pituitary normally lessens its function, and that absence of lesions was coincident with interval when anterior pituitary reaches its maximum concentration. Appropriate organotherapy was therefore instituted (subcutaneous injections of anterior pituitary, etc.) and improvement was prompt and pronounced. This substitutive treatment was later replaced by X-ray stimulation of anterior lobe of pituitary and there has been but one mild recurrence since.

BOOK REVIEWS

Oral Hygiene and the Treatment of Parodontal Diseases. By Russell W. Bunting, D.D.Sc., Professor of Oral Histology and Pathology in the School of Dentistry, University of Michigan, Ann Arbor, Michigan. 187 pp., 80 engravings, 1936.

Bunting's book is notable for its concise presentation of two of the most important subjects in dental practice, "Oral Hygiene" and the "Treatment of Parodontal Diseases."

In his preface the author states that his purpose is to "present certain principles and methods of practice which he has found to be highly effective in the prevention and control of dental and oral diseases" and further, "to call attention of general practitioners of dentistry to the need of a preventive viewpoint in their practice and to the practical means of prevention which lie within their grasp." "Oral Cleanliness and the early treatment or repair of lesions in the hard or soft tissues of the mouth constitute the most effective means of practical preventive dentistry known today."

Part 1., "Oral Hygiene." After a discussion of Oral Sepsis, the author divides his subject into three parts, "Prevention of Dental Caries," "Prevention of Parodontal Diseases," and "Oral Prophylaxis."

As is to be expected, Bunting gives lengthy consideration to the etiology and prevention of dental caries. He presents briefly the outstanding research experiments on caries control by means of diet, including the work of Howe, Hoppert & Weber, Mellanby, Boyd & Drain, Hawkins, Martha Jones, Brodsky, Breese and others, and then, more in detail, his own studies on the significance of Lactobacillus acidophilus in the disease. His data confirms the view that "dental caries is a bacteriologic disease and that L. acidophilus is the most important specific etiologic factor," but it is admitted that the degree of its caries producing activity is determined by other factors, such as dietary considerations, not yet understood.

Nutritional studies were made on varied groups by Koehn & Morrell of the University of Michigan, and the one fairly constant factor seemed to be the correlation of the L. acidophilus findings with the prevalence of caries or comparative freedom from it; further study should be made of all possible methods of inhibiting the growth of this organism in the oral cavity as a means of directly controlling the activity of dental caries. Reviewing the subject, Bunting says, "it will be seen that there are only a few definitely established facts concerning the relationship of diet to dental caries. Positive claims that are made relative thereto are largely unsustained either by the evidence submitted or by the corroboration of others."

"Oral Prophylaxis" in its broadest sense, Bunting states, "consists not only of cleaning the teeth but also includes all measures which tend to protect the mouth tissues from disease and make for oral health." "Mouth cleanliness is at best but a relative matter, for lactic acid fermentations undoubtedly do exist in all mouths to a greater or lesser degree. Our only hope, therefore, in oral prophylaxis, is to reduce these fermentations to the point where the other forces of the mouth will establish caries immunity."

The practice of oral prophylaxis is divided into the following procedures: (1) Removal of calcarious deposits, stains and other extraneous substances from the teeth. (2) Smoothing and polishing of exposed tooth surfaces and rough fillings. (3) Reduction of oral septic processes. (4) Detection and filling of all cavities. (5) Restoration of faulty proximate contacts and tooth contours. (6) Removal of overhanging portions of fillings, crowns, bridges, etc. (7) Relief of undue stresses on the teeth. (8) Instruction of patient as to daily personal care. (9) Sustained periodic supervision and assistance. Each of these processes is

elaborated, and clearly and satisfactorily described.

Part II, "Treatment of Parodontal Diseases." These diseases are subdivided into, Gingivitis, Pyorrhea alveolaris, Pericemental abscess, Vincent's Infection, and Gingival atrophy.

Bunting eschews the terminology made popular by Stillman & McCall's book; used also in Box's writings, and adopted by the American Academy of Periodontology and used by them from 1914 up to the present time; also the nomenclature suggested by the California group of specialists. He elects to use, instead, the term, *parodontal*, in referring to the group of tissues surrounding and supporting the teeth. Were it not that the book for the most part is written in the simplest manner and with the evident (if not avowed) object of making the subject matter comprehensible to novices in the practice of "parodontia" if not beginners in dentistry, this might be understood, since the 1933 report of the nomenclature committee of the A. D. A. endorses (and with some reason), *Paradentium* or *Parodontium* in preference to the popular *Periodontium*. As it is, the various terms employed in Part II, viz. *parodontal tissues, peridental membrane, periodontal fibers, parapical granuloma, periodontal mucosa, pericemental abscess*, when used without explanation, must bewilder the average reader. Their use in such a book is hardly justified by the fact that "more recently the term, Parodontal Diseases (*par*, about, *odont*, tooth) has been used in Europe and to some extent in this country."

Bunting begins the chapter on pyorrhea with a history of our knowledge of the disease recounting the principal theories of its causation and treatment, starting with Fauchard in the 18th century. That he has omitted the recent research in endocrinology and in allerges: and such important names as Becks and Simonton, Kronfeld and Hirschfeld from his book is doubtless a concession to brevity.

Summarizing, Bunting says that regardless of whether vaccines be administered, diets altered, or whatever the therapy employed, success will at best be but temporary unless a careful scaling of the roots be performed and thorough and continuous oral hygiene be established. Here again, Bunting emphasizes the importance of bacteria in oral pathology, saying "it must be recognized that in all cases of pyorrhea, the general bacterial flora of the mouth is abnormal and the first step is to change and decrease the type of the oral flora." The majority of American students "consider pyorrhea to be a local disease arising largely from local irritations and infections," and in Bunting's opinion "the diffuse alveolar type of pyorrhea which Gottlieb describes, and designates as 'genuine pyorrhea' is a form of the disease which may be prevalent in Europe but which is seldom seen in this country." Whether or not this be the case it is unquestionably true, as Bunting states, that for many years a group of men have been successfully treating and controlling pyorrhea by local operative procedures.

Treatment should be attempted only after a careful prognosis has been made, taking account of age, general health and probable coöperation of the patient as well as local clinical findings. Radiograms are not to be relied upon in diagnosis without a careful clinical examination to determine presence and depth of pockets, and vitality or non-vitality of teeth. The greatest success in treatment is to be obtained in early stages of the diseases before extensive destruction has taken place.

Bunting describes in detail the subgingival treatment as well as an interesting "modified surgical method" developed by himself in collaboration with J. W. Kemper at the University of Michigan, by which the diseased interproximal parodontal tissues are removed with a minimum loss of the overlying gingivae. He discusses at some length the relative merits of the conservative and radical methods of treatment.

In his discussion of Vincent's Infection the author's description of the practical clinical procedures leaves nothing to be desired.

The numerous illustrations throughout the book are most practical and helpful; but it is to be regretted that the author did not include among them a few of Box's or Noyes' well nigh classical photomicrographs of the marginal gingivae with their thickened epithelium, and of the gingival crevice, at least. These illustrations might

have served to clarify the author's meaning in the following sentences from the text, (page twenty-three).

> "—of all the mucous coverings of the mouth, the gingivae are most vulnerable to the attack of infectious organisms. In all other portions of the mouth, the mucous membrane with its thickened epithelium offers a continuous protective barrier against the entrance of infectious organisms. But in the process of eruption of the teeth, holes are made in this membrane, one for each tooth, etc."

On the whole, Bunting's "Oral Hygiene and Treatment of Parodontal Diseases" is a very readable book, which by its conciseness, and by its optimistic consideration of the subject of pyorrhea in particular, will commend itself to the busy dentist and should do much to extend the practice of preventive dentistry in all its phases amongst indifferent practitioners.

The assistance of Dr. Dorothy G. Hard of the Department of Dental Hygiene, University of Michigan, is acknowledged by the author.

—*Celia Rich.*

The American Illustrated Medical Dictionary. By W. A. Newman Dorland, A.M., M.D. with the collaboration of E. C. L. Miller, M.D. Seventeenth Edition, revised and enlarged. 1573 pp. 945 illustrations, including 283 portraits. A complete dictionary of the terms used in medicine, surgery, dentistry, pharmacy, chemistry, nursing, veterinary, science, biology, medical biography, etc. The fact that a dictionary, or in fact any other book runs to a seventeenth edition is *prima facie* evidence of the value of the publication.

Whoever possesses this book may have the assurance that he has the latest and best of its kind.

Extract of Review in the J. Am. Dent. A. Sept., 1935.

A Textbook of Physiology. By Wm. D. Zoethout, Ph.D., Professor of Physiology in the Chicago College of Dental Surgery (Loyola University). With 694 pages and 271 illustrations.

As the author states in his preface, the book is intended to "fill a gap between larger texts, . . . and those offering a briefer course." He has admirably done so. The subject is written in a pleasant, readable manner, easy to comprehend, one thought following another and the whole linked in a fine way.

The first, third, fourth, seventh, eighth, twelfth, fourteenth, fifteenth, eighteenth to the twenty-third chapters inclusive should be of special interest to the dentist. Once the subject matter is gone into, the rest of the book will be eagerly read and to good advantage.

Interspersed throughout the book, the author has given practical examples and explanations of the more common ailments. Ordinarily, one would not expect to see a paragraph on Hypertension, Hypotension, or especially on Shock in a Physiology text. Yet, the entire conformity is so logical that it passes as a necessity, with the pleasant feeling one derives from the thought of "so that's it." The more detailed explanations and research data are placed in fine print; to be omitted by the reader if desired. This does not detract from the general work.

On page 454 the author refers to Vitamin D and the teeth. From here he carries on to a more detailed explanation in a later chapter of the influence of the parathyroid hormones on bone and tooth growth. One correlates the other, all woven into an interesting pattern of common sense thought and language.

This invaluable compact book of 694 pages ought to grace every dentist's library. It includes thirty chapters of informative material besides an explanatory ten pages on the contents, a glossary of prefixes and suffixes, an alphabetically arranged glossary of the more technical words and a rich index.

—*S. J. Hodkin, M. D.*

—*Copy from D. Items of Interest. March* 1936.

Vincent's Disease, Pyorrhea and Allied Subjects. By I. Disraeli. 185 pp. 1935

The author tells his confrères in the preface which he dedicates to an expostulation of his own qualifications, that he has compiled statistics based upon extensive research work in periodontology. Then in a second breath he apologetically says that this monograph is not the "last word"—

He receives applause in the Foreword to the book from other colleagues. The reader too, cannot help but applaud the indefatiguable efforts of an author who stayed up nights to burn the "midnight oil" as is attested in the Foreword.

The author has little to say that has not been known by students of periodontology but, he manages to say his piece in a straight-forward manner quite unembellished with any literary flourish.

He says "The author wishes to emphasize the fact that Vincent's Disease today differs greatly from the disease of twenty years ago. The causes of the disease at that time were bacterial, due to improper food and lack of proper living quarters, etc." Then on another page he has a chart showing the organisms of "Vincent's" as he calls them.

In reading this book of variegated topics we had to rub our eyes to determine whether Disraeli was trying to be comical or was he really serious in some statements.

Throughout the entire book there is a trend of implication that dentists do not fulfill their duties to their patients, (that is, all dentists except the author). He says, "Why do dentists so often fail to diagnose incipient pyorrhea—and to inform the patient of the facts?" Also Disraeli states, "In the majority of cases the residual infection, of foreign body such as a bur or a needle is always left in the jaws with the knowledge of the operator."

Right here let us admonish dentists that this book is not to be left in the reception room!

Had this book received serious editing no doubt a good part of its innuendos would have been eliminated together with certain irrelevant chapters. Then it could have been condensed into an excellent article on Vincent's Infection and published in a dental magazine.

The author continually refers to "pyorrhea." The American Academy of Periodontology will be quite irritated when they read this to learn that all their scientific efforts to establish correct nomenclature have not as yet reached some parts of the west coast.

Really, if we have not been enthusiastic about this book, it is Disraeli's own presentation that is at fault and not the well correlated material contained in the text.

—S. J. B.

—*Copy from D. Items of Interest, January, 1936.*

Dental Roentgenology. Second Edition. By LeRoy M. Ennis, D.D.S. assistant Professor of Roentgenology in the Thomas W. Evans Museum, Dental Institute School of Dentistry, University of Pennsylvania, Instructor in Dental Roentgenology, in the Graduate School of Medicine, University of Pennsylvania, Lieutenant-Commander, United States Navy Reserve. 351 pp. 1936.

There appear to be about three times as many American books on this subject as British ones. No British one has been published since 1928, and here we have the second edition of a book originally published in 1931, and which includes new theories and new methods.

A knowledge of electricity is taken for granted, and the author starts at once to describe the construction of the dental X-ray tube, though the Metalix tube so largely used on this side is not mentioned.

Reproduction of the fine detail of dental radiographs is not easy. In the past it has often been a criticism that the reader would find a long description of an illustration in the text and fail to follow it because of poor reproduction. In this book the reproduction of the radiographs is very good and the conditions described in the text can always be seen.

The technique of intra—and extra-oral radiography is dealt with very fully. For intra-oral work the author favors the patient holding the film in the mouth with his finger. For some patients, especially of the rheumatic type, this is not always easy, and there are other and better film holders than the wooden bite-blocks which are the

only type mentioned. In extra-oral radiography the author uses a 2-in. bandage to keep the head in position. Localization and stereoscopic radiographs are well dealt with as is the chemistry of development.

The rest of the book (about half) consists of a description of the normal landmarks of interpretation of radiographs of the mouth and jaw and a description of all pathological conditions. Every condition, normal or abnormal, is well illustrated. (There are 700 illustrations). Generally the descriptions of the various conditions follow the lines with which similar books have familiarized us. Where Ennis differs from previous writers is in his description of nutrient canals of the mandible. C. A. Clark, the pioneer of the subject in Britain, mentions "broad, dark lines often seen between the lower incisors," and states that "their significance is not fully realized, but they are often associated with toxaemia." Ennis says that these are the shadows of branches of the mandibular artery which pass up between the teeth, and, by his radiographs, diagrams, and photographs of dried skulls and decalcified specimens appears to prove this view correct. If this is the case, many a wrong interpretation must have been given in the past. But these lines are not seen in every case. May it not be that in patients of the rheumatic type where there are large cancellous spaces the overlying bone will be thinner and these "lines" more clearly shown?

The book is quite adequate as a textbook both for student and practitioners, but the author realizes that a book alone is not sufficient, that much practical experience is necessary. His warning is worth repeating:—"It cannot be repeated too often that sporadic experiments after a desultory course of reading, coupled, perhaps, with some superficial but high-powered instructions from the equipment salesman will not constitute every dentist a radiologist."

—Copy from The British Dental Journal, May 1, 1936.

Your Breath and Your Health. By Louis M. Pearlman, M.D., Chief of Ear, Nose and Throat Clinic, Harlem Dispensary (1925-1935); Member Vienna A. M. A.; formerly Captain Medical Reserve Corps; Fellow American Medical Association; member New York State and County Medical Societies. Illustrated, 128 pages. Published by the Academy Publishing Company, New York City, 1936.

The importance of a wholesome breath to the dentist cannot be overestimated, and thus this book must make an unusual appeal to the profession. The play on "halitosis" fostered by commercial agencies has no place in a book like this, wherein is presented a serious minded consideration of the nature, cause and effect of bad breath. A clear and satisfactory exposition is made of the conditions leading to bad breath, and the sane and logical treatment of the affections that tend to bring about a departure from the normal, together with the proper procedures for bringing about normality and eliminating the untoward symptoms.

It is in every way a most satisfactory volume, and is not padded to suffocation as many volumes are.

—Copy from J. Am. Dent A., July, 1936.

BOOK NOTICES

Dental Anatomy. A graphic presentation of tooth forms, with an original technique for their reproduction. By M. Diamond, D.D.S., Associate Professor of Dentistry and head of the Department of Dental Anatomy, Columbia University, Dental School. Second Edition. 309 pp., 144 illustrations. 1935.

Practical Dental Anatomy and Tooth Carving. By J. R. Schwartz, D.D.S., Volume 1, illustrated. 233 pp.

Cavity Preparation and Abutment Construction in Bridge Work. By J. R. Schwartz, D.D.S., Volume 2, illustrated. 354 pp.

Practical Orthodontia. By Martin Dewey, D.D.S., F.A.C.D. Revised by G. M. Anderson D.D.S. Fifth revised edition. 502 pp. 1935.

This is actually a new textbook written

by G. M. Anderson, with expansions of various aspects by nine contributors. The plan is a sound one tending to give both unity and completeness. Weinberger introduces the subject by an account of the historical development of Orthodontia which brings forward many interesting facts but is marred by a lack of detachment.

Among the contributed articles are a good and up-to-date chapter on "Tissue Changes Incident to Tooth Movement" (Kronfeld), and an account by Broadbent of his Roentgenographic Cephalometer and the results obtained with it, which are of great interest and promise. A chapter on "Extraction in Relation to Orthodontic Treatment" (Kelsey) written in a spirit of cautious acceptance of the usefulness of extraction, gives but little guidance as to when and where it is of value. The statement is made that "We all know that we can stimulate development by artificial means: indeed this is the basis of orthodontic treatment." This "basis" is one of the assumptions now under question. Rogers writes authoritatively on "Myofunctional Treatment of Malocclusion," but gives no indication of how muscle disorders are to be diagnosed. There is a chapter on Plastic Surgery of the Jaws (Kitlowsky).

In reviewing the author's presentation of Orthodontia as a whole, one has to find a standard of judgment. Only a superman could make a coherent whole of the mass of information which has a bearing on orthodontic practice, but one may reasonably expect that where fairly full knowledge is available it shall be assembled so as to present a picture of definite outlines, and that the major questions awaiting solution shall be clearly stated. In general the author fails to do this. In the most important section of all—the development of the masticatory face—the book is at its weakest. A short chapter on Occlusion touches the fringe of the subject, but the contributions of Ranke, Keith and Campion, Brash, Friel, Korkhaus, and Clinch, with most of Hellman's work, are ignored, though references elsewhere indicate that some of them at least are known to the author. No attempt is made to bring order into the chaos of appliance, the attitude being that "what will give a good result for one patient without discomfort or structural injury may do just the reverse for another," what is successful in one man's hands may be a total failure in another's; without an attempt to find reasons. In fact the book is the book of a broad minded and observant practitioner discoursing on Orthodontia rather than of a teacher presenting a comprehensive, considered view of his subject. As a result it is on the whole easy reading, and many detail points of practical value, which a more formal presentation might omit, find a place; but the defect means that this fifth edition of Practical Orthodontia, though a great advance on previous editions, falls far short of a possible standard.

Illustration and production are excellent, but there is no bibliography.

—*Copy from British Dental Journal, February* 15, 1936.

Twenty-Third Annual Meeting

SAN FRANCISCO, CALIFORNIA

JULY 9, 10, 11, 1936

Thursday

Presentation of Reports

Secretary-Treasurer.....C. H. Gracey
Committee on Program.....C. H. Schott
Committee on Clinics.....C. J. Caraballo
Committee on Nomenclature
A. H. Merritt
Committee on Publication....A. H. Merritt
Committee on Arrangements
J. H. MacKay
President's Address.....Haidee Weeks

Etiologic and Therapeutic Aspects of Paradentosis. Presented by University of California Group—

Newer Knowledge of Paradentosis. Hermann Becks.

Systemic Diseases to be Considered in Paradentosis. Saxton T. Pope.

Nutritional Deficiencies and Their Relation to Paradental Lesions. Nina Simmonds.

Indications and Contra-Indications of Subgingival Curettage in the Treatment of Paradentosis. Dickson Bell.

Significance of Circulatory Channels in Roentgenograms of Alveolar Jaws. W. B. Ryder.

Friday

Report of Committee on Scientific Investigation. Rudolf Kronfeld, Chairman.

The Influence of a Local Excess of Tri-Calcium Phosphate in Controlling Hemorrhage and Healing of Bone Following Injury. J. A. Sinclair, Asheville, N. C.

The Treatment of Paradentosis with Drugs, Instruments and Packs. V. John Oulliber, San Francisco, Calif.

Nutrition and Mal-nutrition from the Laboratory and Clinical Aspects. John A. Marshall, San Francisco.

Results of Experimental Root Canal Therapy. C. M. Dixon, Ann Arbor, Mich.

ROUND TABLE DISCUSSION LUNCHEON

J. D. Towner, Chairman

The Gingival Pocket as a Menace to General Health. John H. McKay, Oakland, Calif.

Care of Interproximal Areas. Dickson G. Bell, San Francisco, Calif.

Gingival Hygiene Requisites. Ruth Brooks Campbell, Los Angeles, Calif.

Drug Therapy in Periodontia. M. Monte Bettman, Portland, Oregon.

The Flap Operation Versus Electro-Coagulation. George P. Kier, San Diego, Calif.

Significance of the Blood Picture in Diagnosis and Treatment of Periclasia. W. Claude Adams, Portland, Oregon.

A Practical Preventive Program in Periodontal Practice. R. W. Rule, San Francisco, Calif.

Advantages of Practice on the Pacific Coast. Mrs. Frederick Bricker, Hollywood, Calif.

CLINIC PROGRAM

C. J. Caraballo, Chairman

Group Clinic

Arranged by Hermann Becks, M. D., D. D. S.

Necessary Requirements for Physical Examination of Paradentic Patients. Saxton T. Pope.

Practical Advice in the Analysis and Evaluation of Nutritional Histories. Nina Simmonds, A. Jensen, R. Collins.

Some Phases of Laboratory Research in the Field of Paradentosis. W. W. Wainwright.

Etiologic and Symptomatologic Classifications of Paradentosis. W. Ryder.

Roentgenographic Interpretation of Fundamental Bone Lesions (Atrophies and Dystrophies) of the Jaws. Hermann Becks.

Newer Investigations of the Problem of Pocket Formation. J. A. Ker.

INDIVIDUAL CLINICS

The Diagnosis of Paradentosis. Willard C. Fleming.

Surgical Eradication. A. W. Ward, San Francisco, Calif.

Unusual Cases (Radiology). George Warner, Denver, Colorado.

Periodontoclasia, My Treatment. Maxwell Dixon, Los Angeles, Calif.

Approaching Periodontosis from the Standpoint of Mineral Metabolism and its Controls. H. L. Spinney, Santa Barbara, Calif.

Visual Field Diagnosis in Dental Foci of Infection. T. A. Brombach, San Francisco, Calif.

Detection and Correction of Occlusal Trauma. L. A. Neil, El Paso, Texas.

Exhibition of Cases of Paradentosis Treated by Conservative Methods Only. J. H. MacKay, San Francisco, Calif.

Developing the Sense of Touch. R. W. Rule, San Francisco, Calif.

ANNUAL BANQUET—Palace Hotel

Saturday

INCIDENTS OF OFFICE PRACTICE

A. H. Merritt, New York, N. Y., Chairman

Method of History Taking in Periodontia. F. A. Bricker, Los Angeles, Calif.

The Treatment of a Stubborn Case of Vincents. J. H. MacKay, San Francisco, Calif.

Treatment of Extensive Periodontoclasia on The Lower Anteriors with some Loose Teeth. R. D. Creamer, Phoenix, Arizonia.

A Case of Reattachment. D. G. Bell, San Francisco, Calif.

Radiology. G. R. Warner, Denver, Colorado.

PRESENTATION OF FELLOWSHIP CERTIFICATES TO:

Rudolf Kronfeld, J. D. Towner